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MEDICO-CHIRURGICAL TRANSACTIONS

PUBLISHED BY

THE ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF
LONDON

VOLUME THE EIGHTIETH

(SECOND SERIES, VOLUME THE SIXTY-SECOND)



LONDON

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September, 1897.



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ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON

PATRON
THE QUEEN

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ELECTED MARCH 1, 1897.

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FREDERICK JAMES GANT
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ROBERT LEAMON BOWLES, M.D.
JOHN MITCHELL BRUCE, M.D.
DAVID FERRIER, M.D.
JOSEPH ARDERNE OMEROD, M.D.
WILLIAM WATSON CHEYNE, F.R.S.
HENRY HUGH CLUTTON, M.B.
FREDERIC S. EVE
WILLIAM APPLETON MEREDITH, C.M.
ROBERT ALEXANDER GIBBONS, M.D.

THE ABOVE FORM THE COUNCIL.

RESIDENT LIBRARIAN
J. Y. W. MAC ALISTER, F.S.A.

PRESIDENTS OF THE SOCIETY FROM ITS FORMATION AS "THE MEDICO-CHIRURGICAL SOCIETY," 1805

ELECTED

- 1806 WILLIAM SAUNDERS, M.D.
- 1808 MATTHEW BAILLIE, M.D.
- 1810 SIR HENRY HALFORD, BART., M.D., G.C.H.
- 1813 SIR GILBERT BLANE, BART., M.D.
- 1815 HENRY CLINE
- 1817 WILLIAM BABINGTON, M.D.
- 1819 SIR ASTLEY PASTON COOPER, BART., K.C.H., D.C.L.
- 1821 JOHN COOKE, M.D.
- 1823 JOHN ABERNETHY
- 1825 GEORGE BIRKBECK, M.D.
- 1827 BENJAMIN TRAVERS
- 1829 PETER MARK ROGET, M.D.
- 1831 SIR WILLIAM LAWRENCE, BART.
- 1833 JOHN ELLIOTSON, M.D. (First President of the Society after
its Incorporation as the Royal Medical and Chirurgical Society of
London in 1834).
- 1835 HENRY EARLE
- 1837 RICHARD BRIGHT, M.D., D.C.L.
- 1839 SIR BENJAMIN COLLINS BRODIE, BART., D.C.L.
- 1841 ROBERT WILLIAMS, M.D.
- 1843 EDWARD STANLEY
- 1845 WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
- 1847 JAMES MONCRIEFF ARNOTT
- 1849 THOMAS ADDISON, M.D.
- 1851 JOSEPH HODGSON
- 1853 JAMES COPLAND, M.D.
- 1855 CÆSAR HENRY HAWKINS
- 1857 SIR CHARLES LOCOCK, BART., M.D.
- 1859 FREDERIC CARPENTER SKEY
- 1861 BENJAMIN GUY BABINGTON, M.D.
- 1863 RICHARD PARTRIDGE
- 1865 SIR JAMES ALDERSON, M.D., D.C.L.
- 1867 SAMUEL SOLLY
- 1869 SIR GEORGE BURROWS, BART., M.D., D.C.L.
- 1871 THOMAS BLIZARD CURLING
- 1873 CHARLES JAMES BLASIUS WILLIAMS, M.D.
- 1875 SIR JAMES PAGET, BART., D.C.L., LL.D.
- 1877 CHARLES WEST, M.D.
- 1879 JOHN ERIC ERICHSEN
- 1881 ANDREW WHYTE BARCLAY, M.D.
- 1882 JOHN MARSHALL
- 1884 SIR GEORGE JOHNSON, M.D.
- 1886 GEORGE DAVID POLLOCK
- 1888 SIR EDWARD HENRY SIEVEKING, M.D., LL.D.
- 1890 TIMOTHY HOLMES
- 1892 SIR ANDREW CLARK, BART., M.D., LL.D., F.R.S.
(*Sir Andrew Clark died 6th November, 1893, and Dr. W. S.
Church, Senior [Medical] Vice-President, officiated as
President until the following 1st March, 1894.*)
- 1894 JONATHAN HUTCHINSON, F.R.S.
- 1896 WILLIAM HOWSHIP DICKINSON, M.D.

HONORARY FELLOWS

(Limited to Twelve.)

Elected

- 1887 FLOWER, SIR WILLIAM HENRY, K.C.B., LL.D., F.R.S.,
Director of the Natural History Department, British
Museum, Cromwell road.
- 1887 FOSTER, MICHAEL, M.D., LL.D., F.R.S., Professor of
Physiology in the University of Cambridge.
- 1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Cor-
responding Member of the Academy of Sciences of
France; The Yews, Reigate Hill, Reigate.
- 1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L.,
LL.D., F.R.S., Corresponding Member of the Academy
of Sciences of France; The Camp, Sunningdale.
- 1896 KELVIN, LORD, D.C.L., F.R.S., Glasgow.
- 1878 LUBBOCK, The Right Hon. SIR JOHN, Bart., M.P., D.C.L.
LL.D., F.R.S., High Elms, Farnborough, Kent, R.S.O.
- 1873 STOKES, SIR GEORGE GABRIEL, Bart., M.A., D.C.L., LL.D.,
F.R.S., Lucasian Professor of Mathematics in the
University of Cambridge; Lensfield Cottage, Cam-
bridge.
- 1887 TURNER, SIR WILLIAM, M.B., D.C.L., LL.D., F.R.S., Pro-
fessor of Anatomy in the University of Edinburgh;
6, Eton Terrace, Edinburgh.

FOREIGN HONORARY FELLOWS

(Limited to Twenty.)

Elected

- 1878 BACCELLI, GUIDO, M.D., Rome.
- 1896 VON BERGMANN, ERNST, Berlin.
- 1887 BILLINGS, JOHN S., M.D., D.C.L.Oxon., New York.
- 1896 CZERNY, VINCENT, M.D., Heidelberg.
- 1896 ERB, WILHELM, M.D., Heidelberg.
- 1887 VON ESMARCH, FRIEDRICH, M.D., Kiel.
- 1896 FOURNIER, ALFRED, M.D., Paris.
- 1896 GERHARDT, CARL, M.D., Berlin.
- 1896 KOCH, ROBERT, M.D., Berlin.
- 1896 KOCHER, THEODORE, M.D., Berne.
- 1868 KÖLLIKER, ALBERT, Würzburg.
- 1896 LAVERAN, A., M.D., Paris.
- 1896 MARIE, PIERRE, M.D., Paris.
- 1896 MITCHELL, SAMUEL WEIR, M.D., Philadelphia.
- 1896 MIRZA-ALI, M.D., Teheran.
- 1856 VIRCHOW, RUDOLPH, M.D., LL.D., Berlin.

FELLOWS

OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF LONDON

EXPLANATION OF THE ABBREVIATIONS

P.—President.	C.—Member of Council.
V.P.—Vice-President.	<i>Sci. Com.</i> —Member of a Scientific Committee.
T.—Treasurer.	<i>Ho. Com.</i> —Member of House Committee.
L.—Hon. Librarian.	<i>Lib. Com.</i> —Member of Library Committee.
S.—Hon. Secretary.	<i>Bldg. Com.</i> —Member of Building Committee.
	<i>Dis. Com.</i> —Member of Discussions Committee.

The abbreviations *Trans.* and *Pro.*, followed by figures, show the number of Papers which have been contributed to the *Transactions* or *Proceedings* by the Fellow whose name they follow. *Referee*, *Sci. Com.*, *Lib. Com.*, *Bldg. Com.*, *Ho. Com.*, and *Dis. Com.*, with the dates of office, are attached to the names of those who have served as Referees of papers and on the Committees of the Society.

Names printed in this *type* are of those Fellows who have paid the Composition Fee in lieu of further annual subscriptions.

Names printed in this *type* are of those Fellows who have paid the Composition Fee entitling them to receive the *Transactions*.

RESIDENT FELLOWS

[N.B.—Fellows are reminded that they are, themselves, responsible for the correctness of the descriptions in the following lists, and it is particularly requested that any change of Title, Appointment, or Residence may be communicated to the Hon. Secretaries before the 1st of July in each year.]

Elected

- 1877 **ABERCROMBIE, JOHN, M.D.**, Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 23, Upper Wimpole street, Cavendish square. C. 1896.—*Trans.* 1.
- 1885 **ABBAHAM, PHINEAS S., M.A., M.D.**, Dermatologist to the West London Hospital, Assistant Surgeon to Hospital for Diseases of the Skin, Blackfriars; 2, Henrietta street, Cavendish square.

Elected

- 1885 AGLAND, THEODORE DYKE, M.D., Physician to St. Thomas's Hospital, and Physician to the Hospital for Consumption and Diseases of the Chest, Brompton ; 74, Brook street, Grosvenor square.
- 1852 ADAMS, WILLIAM, Consulting Surgeon to the Great Northern Central Hospital, the National Hospital for the Paralysed and Epileptic, and the National Orthopædic Hospital ; 7, Loudoun road, St. John's Wood. C. 1873-4. *Trans.* 3.
- 1879 ALLCHIN, WILLIAM HENRY, M.D., F.R.S. Ed., Physician to the Westminster Hospital ; 5, Chandos street, Cavendish square. *Referee*, 1897—.
- 1890 ALLINGHAM, HERBERT WILLIAM, Assistant Surgeon to St. George's Hospital ; 25, Grosvenor street, Grosvenor square.
- 1863 ALTHAUS, JULIUS, M.D., Consulting Physician to the Hospital for Epilepsy and Paralysis, Regent's Park ; 26, Queen Anne street, Cavendish square. *Trans.* 2.
- 1888 ANDERSON, JOHN, M.D., C.I.E., Physician to the Seamen's Hospital, Greenwich ; 9, Harley street, Cavendish square.
- 1890 ANDERSON, WILLIAM, Surgeon to St. Thomas's Hospital ; Professor of Anatomy to the Royal Academy of Arts ; 2, Harley street, Cavendish square. *Lib. Com.* 1896—.
- 1891 ANDREWES, FREDERICK WILLIAM, M.B., Highwood, Hampstead Lane, Highgate.
- 1888 ARKLE, CHARLES, M.D., Assistant Physician to Charing Cross Hospital ; 66, Wimpole street, Cavendish square.
- 1893 BAILEY, ROBERT COZENS, M.S., 21, Welbeck street, Cavendish square.
- 1891 BAKER, CHARLES ERNEST, M.B., 5, Gledhow gardens, South Kensington.
- 1895 BALDWIN, GERALD R., 21, King street, Portman square.
- 1887 BALL, JAMES BARRY, M.D., Physician to the West London Hospital ; 12, Upper Wimpole street, Cavendish square.

Elected

- 1885 **BALLANCE, CHARLES ALFRED, M.S.**, Assistant Surgeon to St. Thomas's Hospital and to the Hospital for Sick Children, Great Ormond street; Surgeon to the National Hospital for the Paralysed and Epileptic, Queen square; 106, Harley street, Cavendish square. *Trans.* 3.
- 1879 **BARKER, ARTHUR EDWARD JAMES**, Professor of the Principles and Practice of Surgery and Professor of Clinical Surgery at University College, and Surgeon to University College Hospital, London; 87, Harley street, Cavendish square. C. 1895-7. *Referee*, 1897—. *Trans.* 7.
- 1876 **BARLOW, THOMAS, M.D., B.S.**, *Trustee for Debenture-holders*; Physician-in-Ordinary to H. M.'s Household; Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond street; 10, Wimpole street, Cavendish square. C. 1892. *Referee*, 1896—. *Trans.* 2.
- 1893 **BARRETT, HOWARD**, 49, Gordon square.
- 1880 **BARROW, A. BOYCE**, Surgeon to King's College Hospital; 37, Wimpole street, Cavendish square.
- 1896 **BARTON, JAMES KINGSTON**, 2, Courtfield road, Gloucester road, South Kensington.
- 1859 **BARWELL, RICHARD**, Consulting Surgeon to the Charing Cross Hospital; 55, Wimpole street. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 12. *Pro.* 1.
- 1868 **BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S.**, Professor of Medicine in University College, London; Physician to University College Hospital and to the National Hospital for the Paralysed and Epileptic; 8A, Manchester square. C. 1885. *Referee*, 1886—96. *Trans.* 2.
- 1890 **BATEMAN, WILLIAM A. F.**, Bridge House, Richmond, Surrey.
- 1891 **BATTEN, FREDERICK E., M.D., B.C.Cantab.**, 124, Harley street.
- 1875 **BEACH, FLETCHER, M.B.**, Winchester House, Kingston Hill [64, Welbeck street].

Elected

- 1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Physician to the City of London Hospital for Diseases of the Chest, and Physician to the Great Northern Central Hospital; 23, Upper Berkeley street.
- 1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-7. *Referee*, 1873-5. *Trans.* 1.
- 1880 BEEVOR, CHARLES EDWARD, M.D., Physician for Out-patients to the National Hospital for the Paralysed and Epileptic, and to the Great Northern Hospital; 33, Harley street, Cavendish square. *Referee*, 1896—. *Trans.* 1
- 1883 BENNETT, STORER, Dental Surgeon to, and Lecturer on Dental Surgery at, the Middlesex Hospital; Dental Surgeon to, and Lecturer on Dental Surgery and Pathology at, the Dental Hospital of London; 17, George street, Hanover square.
- 1877 BENNETT, WILLIAM HENRY, Surgeon to St. George's Hospital; 1, Chesterfield street, Mayfair. C. 1893-4. *Referee*, 1892-93. *Trans.* 4.
- 1897 BERKELEY, COMYNS, M.B., B.C., 53, Wimpole street, W.
- 1845 BERRY, EDWARD UNWIN, 17, Sherrieff road, West Hampstead.
- 1885 BERRY, JAMES, B.S., Demonstrator of Anatomy, St. Bartholomew's Hospital; Surgeon to, and Lecturer on Clinical Surgery at, the Royal Free Hospital; 60, Welbeck street, Cavendish square.
- 1893 BIDWELL, LEONARD A., Senior Assistant Surgeon to the West London Hospital; 59, Wimpole street.
- 1856 Bird, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

Elected

- 1851 **Birkett, JOHN, F.L.S.**, Consulting Surgeon to Guy's Hospital; Corresponding Member of the Société de Chirurgie of Paris; Inspector of Anatomy for the Provinces in England and Wales; 1, Sussex gardens. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869. *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.
- 1881 **Biss, CECIL YATES, M.D.**, Senior Assistant Physician to, and Lecturer on Pharmacology and Therapeutics at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton; 135, Harley street, Cavendish square. *Trans.* 2.
- 1865 **Blandford, GEORGE FIELDING, M.D.**, late Lecturer on Psychological Medicine at St. George's Hospital; 48, Wimpole street, Cavendish square. C. 1883-4.
- 1891 **BOKENHAM, THOMAS JESSOPP**, 10, Devonshire street, Portland place.
- 1890 **BOSTOCK, R. ASHTON**, Surgeon, Scots Guards, 73, Onslow gardens, Brompton.
- 1882 **BOWLBY, ANTHONY ALFRED**, Assistant Surgeon to St. Bartholomew's Hospital; 24, Manchester square. *Trans.* 6.
- 1870 **Bowles, ROBERT LEAMON, M.D.**, 16, Upper Brook street, Grosvenor square. C. 1897—. *Sci. Com.* 1896—. *Trans.* 2.
- 1886 **BOXALL, ROBERT, M.D.**, Assistant Obstetric Physician to, and Lecturer on Practical Midwifery at, the Middlesex Hospital; 29, Weymouth street, Portland place.
- 1884 **BOYD, STANLEY, M.B.**, Surgeon to, and Lecturer on Anatomy at, the Charing Cross Hospital; Surgeon to the Paddington Green Children's Hospital; 134, Harley street, Cavendish square. *Referee*, 1895—. *Trans.* 1.
- 1890 **BRADFORD, JOHN ROSE, M.D., D.Sc., F.R.S.**, Assistant Physician to University College Hospital; 52, Upper Berkeley street, Portman square.

Elected

- 1883 BRADSHAW, JAMES DIXON, M.B., 36, Avenue road, Regent's park.
- 1897 BRAILEY, WILLIAM ARTHUR, M.D., 11, Old Burlington street.
- 1890 BRINTON, ROLAND DANVERS, M.D., 8, Queen's Gate terrace.
- 1868 Broadbent, SIR WILLIAM HENRY, Bart., M.D., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; Physician in Ordinary to H.R.H. the Prince of Wales; 84, Brook street, Grosvenor square. C. 1885. *Referee*, 1881-4, 1891-7. *Trans.* 5.
- 1851 Brodhurst, BERNARD EDWARD, Surgeon to the Royal Orthopædic Hospital and to the Royal Hospital for Incurables; Consulting Surgeon, Belgrave Hospital for Children; Corresponding Member, Société de Chirurgie, Paris, and of the Academy of Sciences, Rome; 21, Portland place, W. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.
- 1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.
- 1880 BROWNE, JAMES WILLIAM, M.B., 160, Holland Park avenue.
- 1881 BROWNE, OSWALD AUCHINLECK, M.A., M.B., Physician to the Royal Hospital for Diseases of the Chest; 43, Bedford square.
- 1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; Physician to the Hospital for Consumption, Brompton; 23, Harley street. C. 1892-7. S. 1893-5. *Sci. Com.* 1889—. *Referee*, 1886-91. *Lib. Com.* 1888-91. *Trans.* 3.

Elected

- 1871 BRUNTON, THOMAS LAUDER, M.D., D.Sc., LL.D., F.R.S., Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; 10, Stratford place, Oxford street. C. 1888-9. *Referee*, 1880-87. *Lib. Com.* 1882-7. *Trans.* 1.
- 1860 BRYANT, THOMAS, Consulting Surgeon to Guy's Hospital; Member of the Société de Chirurgie, Paris; 65, Grosvenor street, Grosvenor square. C. 1873-4. V. P. 1885-6. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 15. *Pro.* 1.
- 1889 BULL, WILLIAM CHARLES, M.B., Aural Surgeon to, and Lecturer on Aural Surgery at, St. George's Hospital; 35, Clarges street, Piccadilly.
- 1893 BURGHARD, FRÉDÉRIC FRANÇOIS, M.D., M.S., Assistant Surgeon to King's College Hospital; 46, Weymouth street, Portland place.
- 1885 BUTLER-SMYTHE, ALBERT CHARLES, Senior Surgeon to the Grosvenor Hospital for Women and Children; 76, Brook street, Grosvenor square.
- 1873 BUTLIN, HENRY TRENTAM, D.C.L., Surgeon to St. Bartholomew's Hospital; 82, Harley street, Cavendish square. C. 1887-8. *Referee*, 1893—. *Trans.* 4. *Pro.* 1.
- 1896 BUTTAR, CHARLES F., M.B., 2, Princes square, Baywater. *Pro.* 1.
- 1883 BUXTON, DUDLEY WILMOT, M.D., B.S., Administrator, and Teacher of the Use, of Anæsthetics, in University College Hospital; Anæsthetist to the National Hospital for the Paralysed and Epileptic, Queen square, and to the London Dental Hospital; 82, Mortimer street, Cavendish square.
- 1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 74, Grosvenor street, Grosvenor square. C. 1885-6. *Referee*, 1887—.

Elected

- 1890 CAGNEY, JAMES, M.A., M.D., in charge of Electrical Department, St. Mary's Hospital; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 101, Harley street, Cavendish square. *Trans.* 1.
- 1885 CAHILL, JOHN, Surgeon to the Hospital of St. John and St. Elizabeth, Great Ormond Street; 12, Seville street, Lowndes square.
- 1893 CALEY, HENRY ALBERT, M.D., Physician in charge of Out-patients and Senior Medical Tutor, St. Mary's Hospital; 24, Upper Berkeley street, Portman square.
- 1887 CALVERT, JAMES, M.D., 36, Queen Anne street, Cavendish square.
- 1897 CANTLIE, JAMES, M.B., 46, Devonshire street.
- 1888 CARLESS, ALBERT, M.S., Assistant Surgeon to King's College Hospital; 10, Welbeck street.
- 1896 CARR, J. WALTER, M.D., Assistant Physician to the Royal Free Hospital; 19, Cavendish place.
- 1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde Park.
- 1853 CARTER, ROBERT BRUDENELL, Consulting Ophthalmic Surgeon to St. George's Hospital; 31, Harley street, Cavendish square. *Trans.* 1.
- 1888 CAUTLEY, EDMUND, M.D., B.C., 15, Upper Brook street.
- 1868 CAVAFF, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. C. 1887. *Referee*, 1896—. *Lib. Com.* 1898—. *Trans.* 1.
- 1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital, Consulting Physician to the London Fever Hospital, and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. C. 1888. *Referee*, 1886-7. *Lib. Com.* 1886-7. *Trans.* 2.
- 1879 CHAMPNEYS, FRANCIS HENRY, M.D., Physician-Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; 42, Upper Brook street, Grosvenor square. *Referee*, 1891—. *Lib. Com.* 1885—. *Trans.* 8.

Elected

- 1868 CHEADLE, WALTER BUTLER, M.D., *Trustee*; Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Hospital for Sick Children; 19, Portman street, Portman square. S. 1886-8. C. 1890-91. *Sci. Com.* 1889-95. *Bldg. Com.* 1889-92. *Referee*, 1885. *Trans.* 1.
- 1879 CHEYNE, WILLIAM WATSON, M.B., F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; 75, Harley street, Cavendish square. C. 1897—. *Referee*, 1894-7. *Lib. Com.* 1886-8, 1891-6. *Trans.* 1.
- 1890 CHILDS, CHRISTOPHER, M.D., 10, Manchester square.
- 1896 CHRISTOPHERSON, JOHN BRIAN, M.B., B.C., 5, Staple Inn.
- 1866 Church, WILLIAM SELBY, M.D., *Hon. Treasurer*, Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. C. 1885-6. V.P. 1892-4. T. 1894—. *Referee*, 1874-81.
- 1879 CLARK, ANDREW, Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 71, Harley street, Cavendish square.
- 1882 CLARKE, ERNEST, M.D., B.S., Surgeon to the Central London Ophthalmic Hospital; Ophthalmic Surgeon to the Miller Hospital; 3, Chandos street, Cavendish square.
- 1890 CLARKE, JAMES JACKSON, M.B., Curator of the Museum and Pathologist to St. Mary's Hospital, 9, Old Cavendish street, Cavendish square.
- 1848 Clarke, JOHN, M.D., 42, Hertford street, Mayfair. C. 1866.
- 1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the West London Hospital; 51, Harley street, Cavendish square. *Trans.* 1.

Elected

- 1879 **Clutton, HENRY HUGH, M.A., M.B.**, Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Victoria Hospital for Children; 2, Portland place. C. 1897—. *Dis. Com.* 1897—. *Referee*, 1896-7. *Trans.* 1.
- 1888 **COCK, FREDERICK WILLIAM, M.D.**, 1, Porchester Houses, Porchester square.
- 1865 **COOPER, ALFRED**, Consulting Surgeon to the West London Hospital; Senior Surgeon to St. Mark's Hospital; 9, Henrietta street, Cavendish square.
- 1889 **COSENS, CHARLES HENRY**, 49, Oxford terrace, Hyde Park.
- 1892 **COTTERELL, EDWARD**, Surgeon for Out-patients, London Lock Hospital; Surgeon to the Cancer Hospital; Surgeon to the West-End Hospital for Epilepsy and Diseases of the Nervous System; 5, West Halkin street, Belgrave square. *Trans.* 1.
- 1860 **Couper, JOHN**, Surgeon to the Royal London Ophthalmic Hospital, and Consulting Surgeon to the London Hospital; 80, Grosvenor street. C. 1876. *Referee*, 1882-3.
- 1877 **COUPLAND, SIDNEY, M.D.**, Physician to, and Joint Lecturer on Practical Medicine at, the Middlesex Hospital; 16, Queen Anne street, Cavendish square. C. 1893-4. *Referee*, 1892-3. *Ho. Com.* 1895—.
- 1862 **Cowell, GEORGE**, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Surgeon to the Victoria Hospital for Children; 19, Harley street, Cavendish square. C. 1882-3.
- 1897 **CRAWFURD, RAYMOND H. PAYNE, M.D.**, 28, Wimpole street.
- 1878 **CRICHTON-BROWNE, SIR JAMES, M.D., LL.D., F.R.S.** Lord Chancellor's Visitor in Lunacy; 61, Carlisle place Mansions, Victoria street.
- 1874 **CRIPPS, WILLIAM HARRISON**, Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. C. 1890-91. *Trans.* 1.

Elected

- 1882 CROCKER, HENRY RADCLIFFE, M.D., Physician to the Skin Department, University College Hospital; late Physician to the East London Hospital for Children; 121, Harley street, Cavendish square. *Trans.* 3.
- 1868 CROFT, JOHN, Consulting Surgeon to St. Thomas's Hospital; 6, Mansfield street, Cavendish square. C. 1884. V.P. 1890. *Referee*, 1885-88. *Lib. Com.* 1877-8. *Trans.* 2.
- 1890 CROWLE, THOMAS HENRY RICKARD, 56, Harley street, Cavendish square.
- 1888 CULLINGWORTH, CHARLES JAMES, M.D., Obstetric Physician and Lecturer on Midwifery at St. Thomas's Hospital; 14, Manchester square. *Referee*, 1896—.
- 1879 CUMBERBATCH, A. ELKIN, M.B., Aural Surgeon to St. Bartholomew's Hospital, and to the National Hospital for the Paralysed and Epileptic; 50, Portland place.
- 1873 CURNOW, JOHN, M.D., Professor of Anatomy in King's College, London, and Physician to King's College Hospital; Senior Physician to the Seamen's Hospital; 35, Welbeck street, Cavendish square. *Referee*, 1884-97.
- 1886 DAKIN, WILLIAM RADFORD, M.D., Obstetric Physician to, and Lecturer in Midwifery at, St. George's Hospital, and Physician to the General Lying-in Hospital; 18, Grosvenor street, Grosvenor square.
- 1872 DALBY, SIR WILLIAM BARTLETT, M.B., Consulting Aural Surgeon to St. George's Hospital; 18, Savile row. C. 1896-7. *Trans.* 3.
- 1891 DALTON, NORMAN, M.D., Physician to King's College Hospital; 4, Mansfield street, Cavendish square.
- 1896 DAUBER, JOHN HENRY, M.A., M.B., B.Ch., L.S.A., 29, Charles street, Berkeley square.
- 1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 36, Harley street, Cavendish square. C. 1892-3. *Referee*, 1890-91. *Trans.* 3.

Elected

- 1889 DEAN, HENRY PERCY, M.S., Assistant Surgeon to the London Hospital; 69, Harley street, Cavendish square.
- 1878 DENT, CLINTON THOMAS, Surgeon to, and Lecturer on Practical Surgery at, St. George's Hospital; 61, Brook street. C. 1890. *Bldg. Com.* 1890-2. *Referee*, 1892—. *Trans.* 4.
- 1891 DE SANTI, PHILIP ROBERT WILLIAM, Assistant Surgeon and Aural Surgeon to the Westminster Hospital; 91, Harley street.
- 1894 DICKINSON, THOMAS VINCENT, M.D., 33, Sloane street.
- 1859 Dickinson, WILLIAM HOWSHIP, M.D., *President*; Consulting Physician to St. George's Hospital, and Consulting Physician to the Hospital for Sick Children; Honorary Fellow of Caius College, Cambridge; 9, Chesterfield street, Mayfair. P. 1896-7. C. 1874-5. V. P. 1887. *Referee*, 1869-73. 1882-6. *Sci. Com.* 1867, 1879, 1889-96. *Trans.* 15.
- 1891 Dickinson, WILLIAM LEE, M.D., Assistant Physician to St. George's Hospital; 9, Chesterfield street, Mayfair.
- 1889 DODD, HENRY WORK, Assistant Surgeon to the Royal Free Hospital, and to the Royal Westminster Ophthalmic Hospital; Ophthalmic Surgeon to the West-End Hospital for Nervous Diseases; 136, Harley street, Cavendish square.
- 1888 DONELAN, JAMES, M.B., M.C., Physician to the Italian Hospital, Queen square; 2, Upper Wimpole street, Cavendish square.
- 1879 DONKIN, HORATIO BRYAN, M.D.Oxon., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 108, Harley street, Cavendish square.
- 1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to the Samaritan Free Hospital; 9, Granville place, Portman square. C. 1893-4. *Lib. Com.* 1891-3. *Trans.* 4.

Elected

- 1867 DOUGLAS-POWELL, SIR RICHARD, Bart., M.D., Physician Extraordinary to H.M. the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 62, Wimpole street, Cavendish square. S. (Oct.) 1883-5. C. 1887-8. *Referee*, 1879-83, 1886. *Trans.* 3.
- 1891 DOVE, PERCY W., "Carshalton," Stapleton Hall Road, Stroud Green.
- 1896 DOWNES, JOSEPH LOCKHART, M.B., C.M., 271, Romford road.
- 1879 DREWITT, F. G. DAWTREY, M.D., Physician to the West London Hospital and to the Victoria Hospital for Children; 2, Manchester square.
- 1893 DRYSDALE, John H., 25, Welbeck street, Cavendish square.
- 1865 DUCKWORTH, SIR DYCE, M.D., LL.D., Hon. Physician to H.R.H. the Prince of Wales; Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Referee*, 1885-97. *Trans.* 2.
- 1876 DUDLEY, WILLIAM LEWIS, M.D., Senior Physician to the City Dispensary; 149, Cromwell road, South Kensington.
- 1871 DUKE, BENJAMIN, M.D., Windmill House, Clapham Common.
- 1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham Common.
- 1884 DUNCAN, WILLIAM, M.D., Obstetric Physician to, and Lecturer on Midwifery at, the Middlesex Hospital; 6, Harley street, Cavendish square.
- 1887 DUNN, HUGH PERCY, Assistant Ophthalmic Surgeon to the West London Hospital; 54, Wimpole street, Cavendish square.
- 1874 DURHAM, FREDERIC, M.B., Senior Surgeon to the North-West London Hospital; late Surgical Registrar to Guy's Hospital; 82, Brook street, Grosvenor square.
- 1894 DURHAM, HERBERT EDWARD, M.B., 82, Brook street, Grosvenor Square. *Trans.* 1.

Elected

- 1868 EASTES, GEORGE, M.B.Lond., 35, Gloucester terrace, Hyde Park. C. 1892-3.
- 1888 ECCLES, ARTHUR SYMONS, M.B., C.M., 23, Hertford street, Mayfair.
- 1893 ECCLES, WILLIAM MCADAM, M.S., 124, Harley street.
- 1891 EDDOWES, ALFRED, M.D., 25, Old Burlington street.
- 1883 EDMUNDS, WALTER, M.C., 75, Lambeth Palace road, Albert Embankment. *Trans.* 3.
- 1884 EDWARDS, FREDERICK SWINFORD, Surgeon to the West London Hospital, and to St. Peter's Hospital for Stone; 55, Harley street, Cavendish square.
- 1879 EVE, FREDERIC S., Surgeon to the London Hospital; Surgeon to the Evelina Hospital for Sick Children; 125, Harley street, Cavendish square. C. 1897—. *Trans.* 2.
- 1877 EWART, WILLIAM, M.D., Physician to St. George's Hospital; 33, Curzon street, Mayfair. C. 1895-7. *Lib. Com.* 1897—. *Sci. Com.* 1889—. *Trans.* 1.
- 1872 FAYRER, SIR JOSEPH, K.C.S.I., LL.D., M.D., F.R.S., Surgeon-General; Honorary Physician to H.M. the Queen, (Military) to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Physician to the Secretary of State for India in Council, and late President of the Medical Board at the India Office; 16, Devonshire street, Portland place. C. 1888. *Referee*, 1881-7.
- 1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882—. *Trans.* 4.
- 1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Neuro-pathology in King's College, London, and Physician to King's College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 34, Cavendish square. *Referee*, 1891-6. C. 1896—. *Dis. Com.* 1896—. *Trans.* 2.

Elected

- 1889 FIELD, GEORGE P., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital, and Dean of the Medical School; 34, Wimpole street, Cavendish square.
- 1866 FITZ-PATRICK, THOMAS, A.M., M.D., 30, Sussex gardens, Hyde Park. C. 1894-6.
- 1891 FLETCHER, HERBERT MORLEY, M.D., 98, Harley street, Cavendish square.
- 1892 FORSBROOK, WILLIAM HENRY RUSSELL, M.D., 139, Buckingham Palace road.
- 1896 FOULERTON, ALEXANDER GRANT RUSSELL, 23, Carlisle mansions, Victoria street, Westminster.
- 1883 FOWLER, JAMES KINGSTON, M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Physician to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.
- 1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician for Skin Diseases to the Westminster Hospital, and to the Skin Department of the Paddington Green Hospital for Children; late Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.
- 1896 FREYER, P. J., Surg.-Lieut.-Colonel, I.M.S., M.A., M.D., 46, Harley street.
- 1884 FULLER, CHARLES CHINNER, 10, St. Andrew's place, Regent's Park.
- 1883 FULLER, HENRY ROXBURGH, M.D., 45, Curzon street, Mayfair.
- 1894 FURNIVALL, PERCY, 34, Adelaide road, South Hampstead.
- 1874 GALABIN, ALFRED LEWIS, M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; 49, Wimpole st., Cavendish square. C. 1892. *Referee*, 1882-91, 1896—. *Lib. Com.* 1883-4. *Trans.* 2.
- 1895 GALLOWAY, JAMES, M.D., 54, Harley street, Cavendish square.
- 1883 GALTON, JOHN CHARLES, M.A., F.L.S., 10, Upper Cheyne row, Chelsea.

Elected

- 1865 **Gant, FREDERICK JAMES**, Consulting Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde Park. C. 1880-81. V.P. 1897—. *Referee*, 1886-97. *Lib. Com.* 1882-5. *Trans.* 3.
- 1854 **Garrod, SIR ALFRED BARING, M.D., F.R.S.**, Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 9.
- 1886 **GARROD, ARCHIBALD EDWARD, M.D.**, Medical Registrar and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Assistant Physician to the Hospital for Sick Children, Great Ormond street; 9, Chandos street, Cavendish square. *Sci. Com.* 1889—. *Lib. Com.* 1896—. *Trans.* 5.
- 1887 **GAY, JOHN**, 119, Upper Richmond road, Putney.
- 1866 **Gee, SAMUEL JONES, M.D., Hon. Librarian; Chairman of Trustees for Debenture-holders**; Physician to St. Bartholomew's Hospital; 31, Upper Brook street, Grosvenor square. C. 1883-4. L. (June) 1887—. *Sci. Com.* 1879. *Bldg. Com.* 1889-92. *Referee*, 1885-7. *Lib. Com.* 1871-6. *Trans.* 1.
- 1885 **GELL, HENRY WILLINGHAM, M.B.**, 36, Hyde Park square.
- 1880 **GIBBONS, ROBERT ALEXANDER, M.D.**, Physician to the Grosvenor Hospital for Women and Children; 29, Cadogan place. C. 1896—. *Trans.* 1.
- 1872 **GILBART-SMITH, THOMAS, M.D.**, Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. C. 1890. *Trans.* 1.
- 1893 **GILES, ARTHUR EDWARD, M.D., B.Sc.**, 58, Harley street.
- 1894 **GILL, RICHARD**, 72, Wimpole street.

Elected

- 1877 GODLEE, RICKMAN JOHN, M.S., *Hon. Librarian* ; Surgeon to University College Hospital, and Professor of Clinical Surgery in University College, London ; Surgeon to the Hospital for Consumption, Brompton ; Consulting Surgeon to the North-Eastern Hospital for Children ; 19, Wimpole street, Cavendish square. S. 1892-4. L. 1895—. *Referee*, 1886-91. *Trans.* 9.
- 1870 GODSON, CLEMENT, M.D., Consulting Physician to the City of London Lying-in Hospital ; 9, Grosvenor street, Grosvenor square.
- 1886 GOLDING-BIRD, CUTHBERT HILTON, M.B., Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital ; 12, Queen Anne street, Cavendish square. *Trans.* 1.
- 1896 GOODALL, EDWARD WILBERFORCE, M.D., B.S., Eastern Hospital, Homerton.
- 1883 GOODHART, JAMES FREDERIC, M.D., Physician to Guy's Hospital ; Consulting Physician to the Evelina Hospital for Sick Children ; 25, Portland place. *Lib. Com.* 1893—.
- 1889 GOODSALL, DAVID HENRY, Surgeon to the Metropolitan Hospital ; Surgeon to St. Mark's Hospital ; 17, Devonshire place, Upper Wimpole street.
- 1895 GOSSAGE, ALFRED MILNE, M.B., 54, Upper Berkeley street.
- 1877 GOULD, ALFRED PEARCE, M.S., Surgeon to the Middlesex Hospital ; 10, Queen Anne street, Cavendish square. C. 1892-3. *Referee*, 1895—. *Ho. Com.* 1892—. *Lib. Com.* 1891. *Trans.* 2.
- 1891 GOW, WILLIAM J., M.D., Assistant Obstetric Physician to St. Mary's Hospital ; Obstetric Physician to the Royal Hospital for Women and Children ; Physician to Out-Patients, Queen Charlotte's Lying-in Hospital ; 27, Weymouth street, Portland place.
- 1873 GOWERS, SIR WILLIAM RICHARD, M.D., F.R.S., Consulting Physician to University College Hospital ; Physician to the National Hospital for the Paralysed and Epileptic ; 50, Queen Anne street, Cavendish square. C. 1891. *Referee* 1888-90. *Lib. Com.* 1884-6. *Trans.* 7.

Elected

- 1892 GRANT, J. DUNDAS, M.A., M.D., 8, Upper Wimpole street, Cavendish square.
- 1868 GREEN, T. HENRY, M.D., Physician to the Charing Cross Hospital, and to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. C. 1886. *Referee*, 1882-5.
- 1885 GRIFFITH, WALTER SPENCER ANDERSON, M.D., Assistant Physician-Accoucheur, St. Bartholomew's Hospital; Physician to Queen Charlotte's Lying-in Hospital; 96, Harley street, Cavendish square.
- 1868 GRIGG, WILLIAM CHAPMAN, M.D., Physician to Queen Charlotte's Lying-in Hospital; Joint Lecturer on Forensic Medicine at the Westminster Hospital Medical School; 27, Curzon street, Mayfair.
- 1889 GUBB, ALFRED S., M.D., 29, Gower street.
- 1883 GUNN, ROBERT MARCUS, M.A., M.B., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to the National Hospital for the Paralysed and Epileptic; Assistant Ophthalmic Surgeon to University College Hospital; 54, Queen Anne street, Cavendish square.
- 1890 GUTHRIE, LEONARD GEORGE, M.D., B.Ch., Physician to the Regent's Park Hospital for Epilepsy and Paralysis; Assistant Physician to the North-West London Hospital; Assistant Physician to the Children's Hospital, Paddington Green; 15, Upper Berkeley street, Portman square.
- 1886 HABERSHON, SAMUEL HERBERT, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 70, Brook street, Grosvenor square.
- 1885 HAIG, ALEXANDER, M.D., Physician to the Metropolitan Hospital, and to the Royal Hospital for Children and Women; 7, Brook street, Grosvenor square. *Trans.* 6.
- 1890 HALE, CHARLES DOUGLAS BOWDICH, M.D., 3, Sussex place, Hyde Park.

Elected

- 1881 **HALL, FRANCIS DE HAVILLAND, M.D.**, Physician to the Westminster Hospital; Physician to St. Mark's Hospital; 47, Wimpole street, Cavendish square. *Referee*, 1893-7.
- 1891 **HAMER, WILLIAM HEATON, M.D.**, 73, Dartmouth Park Hill, Highgate.
- 1889 **HANDFIELD-JONES, MONTAGU, M.D.**, Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Mary's Hospital; Physician to the British Lying-in Hospital; 35, Cavendish square.
- 1856 **Hare, CHARLES JOHN, M.D.**, Consulting Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4. T. 1887-94. V.P. 1894-5. *Bldg. Com.* 1889-92.
- 1864 **Harley, JOHN, M.D., F.L.S.**, Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 9, Stratford place, Oxford street. S. 1875-7. C. 1879-80. V.P. 1895-7. *Referee*, 1871-4, 1882-95. *Sci. Com.* 1879. *Trans.* 10.
- 1893 **HARLEY, VAUGHAN, M.D.**, 25, Harley street, Cavendish square.
- 1892 **HAROLD, JOHN**, 91, Harley street, Cavendish square.
- 1880 **HARRIS, VINCENT DORMER, M.D.**, Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 31, Wimpole street, Cavendish square.
- 1870 **HARRISON, REGINALD**, 6, Lower Berkeley Street, Portman square. C. 1894-5. *Trans.* 2.
- 1870 **HAWARD, J. WARRINGTON, Hon. Treasurer**; Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 16, Savile row, Burlington Gardens. C. 1885. S. 1888-91. V.P. 1894-5. T. (June) 1895—. *Lib. Com.* 1881-4. *Sci. Com.* 1889-91. *Bldg. Com.* (Sec.) 1889-92. *Ho. Com.* 1892—. *Trans.* 2.
- 1891 **HAWKINS, HERBERT PENNELL, M.D., B.C.**, Assistant Physician to St. Thomas's Hospital; 109, Harley street, Cavendish square.

Elected

- 1875 HAYES, THOMAS CRAWFORD, M.A., M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital, and Lecturer on Practical Obstetrics in King's College; Physician for Diseases of Women to the Royal Free Hospital; 17, Clarges street, Piccadilly.
- 1860 HAYWARD, HENRY HOWARD, Consulting Surgeon Dentist to St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.
- 1891 HAYWARD, JOHN ARTHUR, M.D., 58, Brook street. *Pro.* 1.
- 1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. V.P. 1889. *Lib. Com.* 1870-3. *Trans.* 3.
- 1882 HENSLEY, PHILIP JOHN, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square. *Referee*, 1897—.
- 1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 20, Harley street, Cavendish square. *Referee*, 1892—. *Trans.* 1.
- 1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 57, Harley street, Cavendish square.
- 1891 HERRING, HERBERT T., M.B., B.S., 50, Harley street, Cavendish square.
1883. HERRINGHAM, WILMOT PARKER, M.D., Medical Registrar, St. Bartholomew's Hospital; 13, Upper Wimpole street, Cavendish square. *Trans.* 1.
- 1893 HERSCHELL, GEORGE, M.D., 25, Queen Anne street, Cavendish square.

Elected

- 1887 **HEWITT, FREDERIC WILLIAM, M.D.**, Anæsthetist to, and Instructor in Anæsthetics at, the London Hospital; Chloroformist to, and Lecturer on Anæsthetics at, Charing Cross Hospital; Anæsthetist at the Dental Hospital of London; 10, George street, Hanover square. *Trans.* 2.
- 1873 **HIGGENS, CHARLES**, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 52, Brook street, Grosvenor square. C. 1894-5. *Trans.* 2.
- 1890 **HILL, G. WILLIAM, M.D., B.Sc.**, 24, Wimpole street, Cavendish square.
- 1856 **Holmes, TIMOTHY, M.A.**, Consulting Surgeon to St. George's Hospital; Corresponding Member of the Société de Chirurgie, Paris; 6, Sussex place, Hyde Park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. T. 1885-7. P. 1890-92. *Bldg.Com.* (*Chairman*) 1889-92. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5, 1892-5. *Ho. Com.* 1892—. *Trans.* 8.
- 1878 **HOOD, DONALD WILLIAM CHARLES, M.D.**, Senior Physician to the West London Hospital; 43, Green street, Park lane.
- 1883 **HORSLEY, VICTOR ALEXANDER HADEN, F.R.S.**, Surgeon to University College Hospital, Surgeon to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. *Referee*, 1897—. *Trans.* 1.
- 1896 **HORTON-SMITH, PERCIVAL, M.B.**, 15, Upper Brook street. *Sci. Com.* 1897—.
- 1892 **HOWARD, R. J. BLISS, M.D.**, 31, Queen Anne street, Cavendish square.
- 1874 **HOWSE, HENRY GREENWAY, M.S.**, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 59, Brook street, Grosvenor square. C. 1890. *Sci. Com.* 1879. *Referee*, 1887-89. *Trans.* 3.

Elected

- 1889 HUNTER, WILLIAM, M.D., Senior Assistant Physician to the London Fever Hospital; 103, Harley street.
- 1873 HUNTER, SIR W. GUYER, M.D., K.C.M.G., Hon. Surgeon to H.M. the Queen; formerly Principal of, and Professor of Medicine in, Grant Medical College, and Vice-Chancellor of the University, Bombay; Surgeon-General (Retired) Bombay Army; Consulting Physician to Charing Cross Hospital; 21, Norfolk crescent, Hyde Park.
- 1856 HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Senior Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. P. 1894-5. *Referee*, 1876-81, 1883-94. *Lib. Com.* 1864-5. *Trans.* 14. *Pro.* 2.
- 1888 HUTCHINSON, JONATHAN, Jun., Assistant Surgeon to the London Hospital; 1, Park crescent. *Trans.* 1.
- 1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Consulting Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square. C. 1889.
- 1883 JACOBSON, WALTER HAMILTON ACLAND, M.A., M.B., M.Ch., Assistant Surgeon and Lecturer on Anatomy to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 66, Great Cumberland place, Hyde Park. *Referee*, 1895—. *Lib. Com.* 1896—. *Trans.* 2.
- 1892 JAMES, EDWIN MATTHEWS, Belgrave Mansions, Grosvenor gardens.
- 1897 JAMISON, ARTHUR ANDREW, M.D., 18, Lowndes street, Belgrave square.
- 1897 JENNER, LOUIS, M.B., 4A, Bloomsbury square.
- 1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Cancer Hospital, Brompton; 1, Buckingham Palace Mansions.
- 1883 JESSOP, WALTER H. H., M.B., Ophthalmic Surgeon to St. Bartholomew's Hospital; 73, Harley street.

Elected

- 1881 JOHNSON, GEORGE LINDSAY, M.D., Cortina, Netherhall gardens, South Hampstead, and 14, Stratford place, Oxford street.
- 1889 JOHNSON, RAYMOND, M.B., B.S., Assistant Surgeon to University College Hospital; Surgeon to the Victoria Hospital for Children; 20, Weymouth street. *Trans.* 1.
- 1884 JOHNSTON, JAMES, M.D., 53, Prince's square, Bayswater.
- 1887 JONES, HENRY LEWIS, M.D., Medical Officer in charge of Electrical Department at St. Bartholomew's Hospital; 9, Upper Wimpole street, Cavendish square.
- 1896 JONES, L. VERNON, B.A., M.D., B.Ch., 7, Arlington street, St. James's.
- 1881 JULER, HENRY EDWARD, Ophthalmic Surgeon to St. Mary's Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Consulting Ophthalmic Surgeon to the London Lock Hospital; 23, Cavendish square.
- 1893 KANTHACK, ALFRED A., M.D., Lecturer on Pathology, St. Bartholomew's Hospital.
- 1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; 56, Grosvenor street, Grosvenor square.
- 1884 KESER, JEAN SAMUEL, M.D., Physician to the French Hospital; 11, Harley street, Cavendish square.
- 1857 Kiallmark, HENRY WALTER, 5, Pembridge gardens. C. 1890-91.
- 1881 KIDD, PERCY, M.D., Physician to the Hospital for Consumption, Brompton; Assistant Physician to the London Hospital; 60, Brook street, Grosvenor square. *Trans.* 4.
- 1851 Kingdon, JOHN ABERNETHY, Consulting Surgeon to the Bank of England; 2, Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.
- 1896 LANE, JAMES ERNEST, 46, Queen Anne street, Cavendish square.

Elected

- 1884 LANE, WILLIAM ARBUTHNOT, M.S., Lecturer on Anatomy at Guy's Hospital; Assistant Surgeon to the Hospital for Sick Children; 21, Cavendish square. *Trans.* 4.
- 1882 LANG, WILLIAM, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 22, Cavendish square.
- 1894 LANGDON-DOWN, REGINALD LANGDON, M.B., B.C., 81, Harley street.
- 1865 Langton, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 62, Harley street, Cavendish square. C. 1881-2. V.P. 1895-7. *Referee*, 1885-95. *Lib. Com.* 1879-80, 1888-95. *Trans.* 2.
- 1890 LAW, EDWARD, M.D., C.M., 35, Harley street, Cavendish square.
- 1888 LAWRENCE, LAURIE ASHER, 125, Harley street, Cavendish square.
- 1893 LAWSON, ARNOLD, 12, Harley street, Cavendish square.
- 1884 LAWSON, GEORGE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Surgeon to the Royal London Ophthalmic Hospital; Surgeon to the Middlesex Hospital; 12, Harley street, Cavendish square.
- 1892 LEADAM, WILLIAM WARD, M.D., 80, Gloucester terrace, Hyde Park.
- 1843 Lee, HENRY, Consulting Surgeon to St. George's Hospital; 61, Queenborough terrace, Hyde Park. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 14. *Pro.* 2.
- 1895 LEES, DAVID BRIDGE, M.D., 22, Weymouth street, Portland place.
- 1895 LESLIE, ROBERT MURRAY, M.B., 58, Harley street, Cavendish square.

Elected

- 1886 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., Obstetric Physician to the London Hospital ; 72, Harley street, Cavendish square. *Trans.* 1.
- 1896 LEWIS, FREDERICK HENRY, M.B., St. Bartholomew's Hospital, and 71, The Drive, West Brighton.
- 1878 LISTER, LORD, D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen ; Emeritus Professor of Clinical Surgery in King's College, London ; and Consulting Surgeon to King's College Hospital ; 12, Park crescent, Regent's Park. C. 1892.
- 1891 LITTLE, ERNEST MUIRHEAD, 40, Seymour street, Portman square.
- 1889 LITTLE, JOHN FLETCHER, M.B., 32, Harley street, Cavendish square.
- 1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Central Hospital ; Assistant Surgeon to, and Demonstrator of Operative Surgery at, St. Bartholomew's Hospital ; 19, Upper Berkeley street, Portman square. *Trans.* 4.
- 1881 LUCAS, RICHARD CLEMENT, B.S., M.B., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital ; Surgeon to the Evelina Hospital for Sick Children ; Corresponding Member of the Société de Chirurgie of Paris ; 50, Wimpole street, Cavendish square. *Trans.* 2.
- 1888 LUFF, ARTHUR PEARSON, M.D., B.Sc., Physician to Outpatients and Lecturer on Medical Jurisprudence at St. Mary's Hospital ; Official Analyst to the Home Office ; 31, Weymouth street, Portland place.
- 1887 LUSH, PERCY J. F., M.B., 4, Maresfield gardens, Hampstead.
- 1873 MacCarthy, JEREMIAH, M.A., Surgeon to the London Hospital, late Lecturer on Surgery at the London Hospital Medical College ; 1, Cambridge place, Victoria road, Kensington. C. 1886-7. *Lib. Com.* 1882-5. *Referee*, 1890—.

Elected

- 1867 **MAC CORMAC, SIR WILLIAM, Bart., M.A., M.Ch., D.Sc.,** Consulting Surgeon to St. Thomas's Hospital; 13, Harley street. C. 1884-5. V.P. 1896-7. *Referee*, 1889-96. *Lib. Com.* 1895. *Trans.* 1.
- 1894 **MAOPADYEN, ALLAN, M.D., B.S.,** 101, Great Russell street.
- 1896 **MACGREGOR, ALEXANDER, M.D.,** 7, Harley street.
- 1880 **McHARDY, MALCOLM MACDONALD,** Ophthalmic Surgeon to King's College Hospital, and Professor of Ophthalmic Surgery in King's College, London; Surgeon to the Royal Eye Hospital, Southwark; 5, Savile row.
- 1873 **MACKELLAR, ALEXANDER OBERLIN, M.Ch.,** Surgeon to St. Thomas's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 79, Wimpole street, Cavendish square.
- 1881 **MACKENZIE, STEPHEN, M.D.,** Physician to the London Hospital, and Lecturer on the Principles and Practice of Medicine at the London Hospital Medical College; Physician to the Royal London Ophthalmic Hospital; 18, Cavendish square. *Referee*, 1890—. *Trans.* 1.
- 1879 **MACLAGAN, THOMAS JOHN, M.D.,** Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.
- 1889 **MACLEHOSE, NORMAN MACMILLAN, M.B., C.M.,** 13, Queen Anne street, Cavendish square.
- 1893 **MCLEOD, KENNETH, M.D.,** 39, Clanricarde gardens, Bayswater. *Trans.* 1.
- 1876 **MACNAMARA, N. CHARLES,** Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Consulting Surgeon to the Royal Westminster Ophthalmic Hospital; 13, Grosvenor street. C. 1891-2. *Referee*, 1884-90, 1895-7. *Lib. Com.* 1886-90.
- 1881 **MACREADY, JONATHAN FORSTER CHRISTIAN HORACE,** Surgeon to the Great Northern Hospital; 132, Harley street, Cavendish square.
- 1880 **MADDICK, EDMUND DISTIN,** 2, Chandos street, Cavendish square.

Elected

- 1886 **MAGUIRE, ROBERT, M.D.**, Physician to Out-patients and Joint Lecturer on Pathology at St. Mary's Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 4, Seymour street, Portman square. *Sci. Com.* 1889—.
- 1880 **MAKINS, GEORGE HENRY**, Assistant Surgeon to St. Thomas's Hospital; Surgeon to the Evelina Hospital for Children; 47, Charles street, Berkeley square. *Trans.* 1.
- 1885 **MALCOLM, JOHN DAVID, M.B., C.M.**, Surgeon to the Samaritan Free Hospital; 13, Portman street, Portman square. *Trans.* 2.
- 1890 **MANSON, PATRICK, M.D., C.M., LL.D.**, Physician to the Seamen's Hospital, Greenwich; 21, Queen Anne street, Cavendish square.
- 1855 **MARCEY, WILLIAM, M.D., F.R.S.**, Flowermead, Wimbledon Park. C. 1871. V.P. 1897—. *Referee*, 1866-70, 1883-6. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.
- 1867 **MARSH, F. HOWARD**, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 30, Bruton street, Berkeley square. C. 1882-3, 1889. S. 1885-7. V.P. 1891-3. *Lib. Com.* 1880-1. *Trans.* 4.
- 1891 **MARTIN, HENRY CHARRINGTON, M.D.**, 27, Oxford square.
- 1884 **MARTIN, SIDNEY HARRIS COX, M.D., F.R.S.**, Assistant Physician to University College Hospital, and to the Hospital for Consumption, Brompton; Professor of Pathology, University College, London; 10, Mansfield street, Portland place.
- 1892 **MASTERS, JOHN ALFRED, M.D.**, 57, Lexham gardens, Kensington.
- 1891 **MAY, WILLIAM PAGE, M.D., B.Sc.**, 49, Welbeck street.
- 1891 **MERCIER, CHARLES ARTHUR, M.B.**, Lecturer on Neurology and Insanity to Westminster Hospital; 8, New Court, Lincoln's Inn, and Flower House, Southend, Catford.

Elected

- 1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 21, Manchester square. C. 1897—. *Trans.* 1.
- 1897 MERRY, WILLIAM JOSEPH COLLINGS, M.D., B.Ch., 1, Cleveland square, Hyde park.
- 1894 MICHELS, ERNST, M.D., 6, West street, Finsbury circus. *Trans.* 2.
- 1893 MILEY, MILES, M.B., 21, Belsize avenue, Hampstead.
- 1891 MOLINE, PAUL, M.B., 42, Walton street, Chelsea.
- 1873 MOORE, NORMAN, M.D., *Hon. Secretary*, Assistant Physician and Lecturer on Medicine to St. Bartholomew's Hospital; 94, Gloucester place, Portman square. C. 1891-2. S. 1896—. *Referee*, 1886-90. *Sci. Com.* 1889—.
- 1878 MORGAN, JOHN HAMMOND, M.A., Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street; 68, Grosvenor street. C. 1895-7. *Dis. Com.* 1896-7. *Trans.* 2.
- 1894 MORISON, ALEXANDER, M.D., 14, Upper Berkeley street, W.
- 1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 8, Cavendish square. C. 1888-9. *Referee*, 1882-7. *Lib. Com.* 1895-6. *Trans.* 10.
- 1879 MORRIS, MALCOLM ALEXANDER, Surgeon to the Skin Department of, and Lecturer on Dermatology at, St. Mary's Hospital; 8, Harley street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.
- 1885 MOTT, FREDERICK WALKER, M.D., Assistant Physician to Charing Cross Hospital; Pathologist to the London County Council; 84, Wimpole street, Cavendish square.
- 1896 MURPHY, JAMES KEOGH, M.B., 53, Princes street, Bayswater.
- 1888 MURRAY, HUBERT MONTAGUE, M.D., Physician to Outpatients, and Lecturer on Pathology at, the Charing Cross Hospital; 27, Savile row.

Elected

- 1880 MURRELL, WILLIAM, M.D., Physician to Out-patients, and Lecturer on Materia Medica and Therapeutics, at the Westminster Hospital; 17, Welbeck street, Cavendish square. *Sci. Com.* 1889—. *Trans.* 1.
- 1892 MYDDELTON-GAVEY, E. HERBERT, 124, Harley street, Cavendish square.
- 1863 MYERS, ARTHUR BOWEN RICHARDS, late Brigade-Surgeon, Brigade of Guards; 43, Gloucester street, Warwick square. *C.* 1878-9. *Lib. Com.* 1877.
- 1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 5, Wimpole street, Cavendish square. *Referee*, 1892—.
- 1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.
- 1880 OGILVIE, GEORGE, M.B., B.Sc., Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; 22, Welbeck street, Cavendish square. *Trans.* 1.
- 1880 OGILVIE, LESLIE, M.B., B.Sc., Physician to the Paddington Green Children's Hospital; 46, Welbeck street, Cavendish square.
- 1891 OGLE, CYRIL, M.A., M.B., 96, Gloucester place.
- 1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 96, Gloucester place, Portman square. *C.* 1873. *V.P.* 1886. *Referee*, 1864-72. *Trans.* 4.
- 1860 OGLE, WILLIAM, M.D., late Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. *S.* 1868-70. *C.* 1876-7. *V.P.* 1887. *Lib. Com.* 1871-5. *Trans.* 5.
- 1896 OLIVER, GEORGE, M.D., 77, Wimpole street, Cavendish square.
- 1892 OPENSHAW, T. HORROCKS, M.B., M.S., Assistant Surgeon to, and Lecturer on Anatomy at, the London Hospital; 16, Wimpole street, Cavendish square.

Elected

- 1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 37, Upper Brook street, Grosvenor square. C. 1889-90. *Sci. Com.* 1889—. *Referee*, 1884-8. *Trans.* 6.
- 1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Physician to St. Bartholomew's Hospital; Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 25, Upper Wimpole street. C. 1897—. *Lib. Com.* 1896—. *Trans.* 1.
- 1875 OSBORN, SAMUEL C., 10, Maddox street, Regent street, and Maisonnnette, Datchet, Berks.
- 1879 OWEN, EDMUND, M.B., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; Senior Surgeon to the Hospital for Sick Children, Great Ormond street; 64, Great Cumberland place, Hyde park. C. 1896-7. *Trans.* 3.
- 1882 OWEN, HERBERT ISAMBARD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. George's Hospital; 40, Curzon street, Mayfair. *Bldg. Com.* 1889-92. *Referee*, 1893, 1895—.
- 1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. C. 1890-91. *Referee*, 1884-89. *Lib. Com.* 1886-8. *Trans.* 4.
- 1840 PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Foreign Associate of the Académie de Médecine, Paris; 5, Park square West, Regent's park. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.
- 1886 PAGET, STEPHEN, Surgeon to, and Surgeon to the Aural Department at, the West London Hospital; 57, Wimpole street, Cavendish square.

Elected

- 1895 PARKER, CHARLES ARTHUR, 41, Queen Anne street, Cavendish square.
- 1873 PARKER, ROBERT WILLIAM, *Hon. Secretary*; Senior Surgeon to the East London Hospital for Children; Senior Surgeon to the German Hospital; 13, Welbeck street, Cavendish square. C. 1888-9. S. 1895—. *Bldg. Com.* 1889-92. *Referee*, 1891-5. *Lib. Com.* 1885-87, 1892-5. *Ho. Com.* 1892-5. *Trans.* 4.
- 1889 PARSONS, J. INGLIS, M.D., Physician to Out-patients, Chelsea Hospital for Women; 3, Queen street, Mayfair.
- 1883 PASTEUR, WILLIAM, M.D., Assistant Physician to the Middlesex Hospital; Physician to the North-Eastern Hospital for Children; 4, Chandos street, Cavendish square.
- 1891 PATERSON, WILLIAM BROMFIELD, 64, Brook street, Grosvenor square.
- 1891 PATON, EDWARD PERCY, M.D., 84, Park street, Grosvenor square.
- 1865 PAVY, FREDERICK WILLIAM, M.D., LL.D., F.R.S., Consulting Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. V.P. 1893-4. *Referee*, 1871-82. *Trans.* 1.
- 1869 PAYNE, JOSEPH FRANK, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. C. 1887. *Referee*, 1890—. *Sci. Com.* 1879. *Lib. Com.* 1878-85, 1889—.
- 1894 PEGLER, L. HEMINGTON, M.D., 25, Old Burlington street.
- 1887 PENROSE, FRANCIS GEORGE, M.D., Assistant Physician to St. George's Hospital; 4, Harley street, Cavendish square. *Sci. Com.* 1889—.
- 1897 PERRAM, CHARLES HERBERT, M.D.
- 1890 PERRY, EDWIN COOPER, M.D., Assistant Physician to, and Demonstrator of Pathology at, Guy's Hospital; The College, Guy's Hospital.
- 1895 PHEAR, ARTHUR G, M.D., Assistant Physician and Pathologist to the Metropolitan Hospital, 13, Welbeck street, Cavendish square.

Elected

- 1883 PHILLIPS, CHARLES DOUGLAS F., M.D., LL.D., 10, Henrietta street, Cavendish square.
- 1884 PHILLIPS, GEORGE RICHARD TURNER, 24, Palace Court, Notting hill gate.
- 1888 PHILLIPS, JOHN, M.D., Assistant Obstetric Physician, King's College Hospital; Physician to the British Lying-in Hospital; 68, Brook street, Grosvenor square. *Trans.* 1.
- 1889 PHILLIPS, SIDNEY, M.D., Physician and Lecturer on Medicine at St. Mary's Hospital; Senior Physician to the London Fever Hospital, and to the Lock Hospital; 62, Upper Berkeley street, Portman square.
- 1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884-5. V.P. 1893-4. *Referee*, 1882-3, 1891-93. *Sci. Com.* 1870, 1889—. *Lib. Com.* 1879-81.
- 1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to, and Pathologist at, Guy's Hospital; 15, Portland place. *Trans.* 1. *Referee*, 1897—.
- 1889 PITTS, BERNARD, M.B., M.C., Surgeon to St. Thomas's Hospital; 109, Harley street, Cavendish square. *Referee*, 1897—.
- 1885 POLAND, JOHN, Surgeon to the Miller Hospital, Greenwich; 4, St. Thomas's street, Southwark.
- 1884 POLLARD, BILTON, B.S., Surgeon to University College Hospital, Surgeon to the North-Eastern Hospital for Children; 24, Harley street, Cavendish square. *Trans.* 1.
- 1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. V.P. 1896-7. *Referee*, 1872-81.
- 1894 POLLOCK, WILLIAM RIVERS, M.B., B.C., 56, Park street, Grosvenor square.

Elected

- 1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women, Waterloo road; 32, Wimpole street, Cavendish square. C. 1890-91. *Referee*, 1887-9, 1892—. *Lib. Com.* 1895—. *Trans.* 2.
- 1885 PORT, HEINRICH, M.D., Physician to the German Hospital; 48, Finsbury square.
- 1887 POWER, D'ARCY, M.A., M.B., Surgeon to the Victoria Hospital for Children; 10A, Chandos street, Cavendish Square. *Lib. Com.* 1896—. *Trans.* 2.
- 1867 Power, HENRY, Consulting Ophthalmic Surgeon to St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde Park. C. 1882-3. V.P. 1892-3. *Referee*, 1870-81, 1891—, *Sci. Com.* 1870, 1889—. *Lib. Com.* 1872-8.
- 1857 Priestley, SIR WILLIAM OVEREND, M.D., LL.D., M.P., Consulting Physician to King's College Hospital, and to the West London Hospital and the British Lying-in Hospital; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884-5. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.
- 1883 PRINGLE, JOHN JAMES, M.B., C.M., Lecturer on Practical Medicine, Physician in Charge of Skin Department at, the Middlesex Hospital; 23, Lower Seymour street, Portman square. *Trans.* 2.
- 1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.
- 1877 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Member of the Senate of the University of London; 48, Brook street, Grosvenor square. C. 1893-4. *Lib. Com.* 1887-93. *Referee*, 1897—. *Trans.* 1.

Elected

- 1850 **Quain, SIR RICHARD, Bart., M.D., (Hon.) M.D.Dublin, LL.D.Ed., F.R.S.,** Physician Extraordinary to H.M. the Queen; President of the General Medical Council; Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.
- 1893 **RANKIN, GUTHRIE, 4, Chesham street, Belgrave square.**
- 1892 **RAYNER, HENRY, M.D., 2, Harley street, Cavendish square.**
- 1869 **READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.**
- 1891 **REECE, RICHARD JAMES, 31, Holland Villas road.**
- 1891 **REMFRY, LEONARD, M.D.,** Obstetrical Physician to the Great Northern Central Hospital; Assistant Obstetric Physician to, and Assistant Lecturer on Obstetric Medicine at, St. George's Hospital; 60, Great Cumberland place.
- 1891 **RENDEL, ARTHUR BOWEN, M.A., M.B., B.C., 43, Albion street, Hyde Park.**
- 1887 **RICHARDSON, GILBERT, M.A., M.D.,** Hawthorn House, Putney.
- 1863 **RINGER, SYDNEY, M.D., F.R.S.,** Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80, 1889-97. *Trans.* 6.
- 1896 **ROBINSON, HENRY BETHAM, 1, Upper Wimpole street.**
- 1896 **ROBERTS, CHARLES HUBERT, M.D., 21, Welbeck street.**
- 1893 **ROBERTS, D. WATKIN, M.D., 56, Manchester street, Manchester square.**
- 1878 **ROBERTS, FREDERICK THOMAS, M.D.,** Professor of Materia Medica and Therapeutics, and of Clinical Medicine, in University College, London; Physician to University College Hospital; Consulting Physician to the Hospital for Consumption, Brompton; 102, Harley street, Cavendish square. C. 1894-5. *Sci. Com.* 1889—.

Elected

- 1889 **ROBERTS, SIR WILLIAM, M.D., B.A., F.R.S.**, 8, Manchester square. *Referee*, 1897—. *Trans.* 2.
- 1890 **ROLLESTON, HUMPHRY DAVY, M.D.**, Assistant Physician to, and Lecturer on Pathology at, St. George's Hospital; 112, Harley street, Cavendish square.
- 1857 **Rose, HENRY COOPER, M.D.**, Consulting Surgeon to the Hampstead Dispensary; 16, Warwick road, Maida Vale. C. 1886-7. *Trans.* 1.
- 1883 **ROSE, WILLIAM, M.B.**, Professor of Clinical Surgery in King's College; Surgeon to King's College Hospital; and Consulting Surgeon to the Royal Free Hospital; 17, Harley street, Cavendish square.
- 1888 **ROUGHTON, EDMUND WILKINSON, B.S., M.D.**, Warden of the College of St. Mary's Hospital; Assistant Surgeon to the Royal Free Hospital; 33, Westbourne terrace, Hyde Park. *Trans.* 1.
- 1882 **ROUTH, AMAND JULES McCONNEL, M.D., B.S.**, Obstetric Physician to Out-patients, and Lecturer on Practical Midwifery, at the Charing Cross Hospital; Physician to the Samaritan Free Hospital for Women and Children; 14A, Manchester square.
- 1849 **Routh, CHARLES HENRY FELIX, M.D.**, Consulting Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.
- 1891 **RUSSELL, J. S. RISIEN, M.B., C.M.**, Assistant Physician to the Metropolitan Hospital; 4, Queen Anne street, Cavendish square.
- 1886 **SAINSBURY, HARRINGTON, M.D.**, Physician to the Royal Free Hospital; Assistant Physician to the City of London Hospital for Diseases of the Chest; 63, Welbeck street, Cavendish square. *Trans.* 1.
- 1869 **Sansom, ARTHUR ERNEST, M.D.**, Physician to the London Hospital; Consulting Physician; North - Eastern Hospital for Children; 84, Harley street, Cavendish square. C. 1887-8. *Referee*, 1889—. *Trans.* 3.

Elected

- 1845 **SAUNDERS, SIR EDWIN**, Surgeon-Dentist to H.M. the Queen, and to their R.H. the Prince and Princess of Wales; Fairlawn, Wimbledon Common. C. 1872-3.
- 1879 **SAVAGE, GEORGE HENRY, M.D.**, Lecturer on Mental Diseases at Guy's Hospital; 3, Henrietta street, Cavendish square.
- 1883 **SCHÄFER, EDWARD ALBERT, F.R.S.**, Jodrell Professor of Physiology, University College, London; University College, Gower street. *Referee*, 1888—. *Sci. Com.* 1889—.
- 1892 **SCHORSTEIN, GUSTAVE, M.A., M.B., B.Ch., D.P.H.**, Assistant Physician to the London Hospital, and to the Hospital for Consumption, Brompton; 11, Portland place.
- 1887 **SCOTT, HARRY, M.D.**, 27, St. Ermin's mansions, Westminster.
- 1882 **SCRIVEN, JOHN BARCLAY**, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.
- 1863 **Sedgwick, WILLIAM**, 101, Gloucester place, Portman square. C. 1884-5. *Trans.* 3.
- 1892 **DE SEGUNDO, CHARLES SEMPILL**, 6, Brook street, Hanover square.
- 1892 **SELWYN-HARVEY, JOHN STEPHENSON, M.D.**, 1, Astwood road, Cromwell road.
- 1877 **SEMON, SIR FELIX, M.D.**, Physician for Diseases of the Throat to St. Thomas's Hospital; 39, Wimpole street, Cavendish square. C. 1895-7. *Lib. Com.* 1894-5. *Trans.* 3.
- 1894 **SEWILL, JOSEPH SEFTON**, 9A, Cavendish square.
- 1882 **SHARKEY, SEYMOUR JOHN, M.D.**, Physician to, and Joint Lecturer on Medicine at, St. Thomas's Hospital; 22, Harley street, Cavendish square. *Referee*, 1897—. *Trans.* 2.
- 1886 **SHAW, LAURISTON ELGIE, M.D.**, Assistant Physician to Guy's Hospital; 10, St. Thomas's street, Southwark.

Elected

- 1884 SHEILD, ARTHUR MARMADUKE, M.B., B.C., Assistant Surgeon to St. George's Hospital; 4, Cavendish place. *Referee*, 1897—. *Trans.* 4.
- 1896 SHORE, THOMAS WILLIAM, M.D., The Warden's House, St. Bartholomew's Hospital.
- 1893 SIBLEY, WALTER KNOWSLEY, M.D., B.C., Senior Physician to Out-patients, North-West London Hospital; 7, Upper Brook street.
- 1848 SIEVEKING, SIR EDWARD HENRY, M.D., LL.D., F.S.A. Physician-in-Ordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's and the Lock Hospitals; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. P. 1888-9. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.
- 1886 SILCOCK, ARTHUR QUAREY, B.S., Surgeon in charge of Out-patients, St. Mary's Hospital; Surgeon to the Royal London Ophthalmic Hospital; 52, Harley street, Cavendish square. *Lib. Com.* 1895—.
- 1842 SIMON, SIR JOHN, K.C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee*, 1851-3, 1866-81. *Trans.* 1.
- 1892 SIMS, FRANCIS MANLEY BOLDERO, 12, Hertford street, Mayfair.
- 1893 SISLEY, RICHARD, M.D., 14, Park lane.
- 1894 SLATER, CHARLES, M.B., 81, St. Ermin's mansions, Westminster.
- 1896 SLOANE, JOHN STRETTON, M.B., 3, Montague mansions, Portman square.
- 1890 SMALE, MORTON, 22A, Cavendish square.
- 1879 SMITH, E. NOBLE, Surgeon to All Saints' Children's Hospital; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

Elected

- 1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 15, Queen Anne street, Cavendish square.
- 1891 SMITH, G. COCKBURN, M.D., 5, Inverness gardens, Kensington.
- 1838 SMITH, HENRY SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.
- 1866 SMITH, HEYWOOD, M.D., 18, Harley street, Cavendish square.
- 1889 SMITH, ROBERT PERCY, M.D., B.S., Resident Physician and Medical Superintendent, Bethlem Royal Hospital, St. George's road, Southwark.
- 1892 SMITH, SOLOMON CHARLES, M.D., Consulting Surgeon to the Royal Halifax Infirmary, and Physician to the Westminster General Dispensary, 1, Montague Mansions, Portman square.
- 1863 SMITH, SIR THOMAS, Bart., Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. V.P. 1887-8. *Referee*, 1873-4, 1880-6. *Sci. Com.* 1867. *Trans.* 4.
- 1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital Society, Greenwich.
- 1874 SMITH, WILLIAM ROBERT, M.D., D.Sc., Barrister-at-Law, Professor of Forensic Medicine in, and Director of the Laboratories of State Medicine at, King's College, London; Medical Officer to the School Board for London; 74, Great Russell Street. *Trans.* 1.
- 1865 Southey, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

Elected

- 1889 SPENCER, HERBERT R., M.D., B.S., Professor of Midwifery in University College; Obstetric Physician to University College Hospital; 10, Mansfield street, Portland place. *Referee*, 1894—.
- 1887 SPENCER, WALTER GEORGE, M.B., M.S., Assistant Surgeon to the Westminster Hospital; 35, Brook street, Grosvenor square. *Trans.* 2.
- 1888 SPICER, ROBERT HENRY SCANES, M.D., Physician to the Department for Diseases of the Throat, St. Mary's Hospital; 28, Welbeck street, Cavendish square.
- 1890 SPICER, WILLIAM THOMAS HOLMES, M.B., 47, Welbeck street, Cavendish square.
- 1875 SPITTA, EDMUND JOHNSON, Ivy House, Clapham Common, Surrey.
- 1851 Spitta, ROBERT JOHN, M.D., 2, The Sweep, East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.
- 1885 SQUIRE, JOHN EDWARD, M.D., Physician to the North London Hospital for Consumption; 122, Harley street, Cavendish square. *Trans.* 2.
- 1897 STAINER, EDWARD, M.A., M.B., 2, Vernon Chambers, Bloomsbury.
- 1896 STEPHENS, JOHN WILLIAM WATSON, M.B., B.C., 25, East Paul's Wharf.
- 1856 Stoker, ALONZO HENRY, M.D., Peckham House, Peckham.
- 1884 STONHAM, CHARLES, Surgeon to the Westminster Hospital, and Curator of Anatomical Museum; 4, Harley street, Cavendish square.
- 1896 SUTHERLAND, GEORGE ALEXANDER, M.D., 9, Old Cavendish street.
- 1871 Sutherland, HENRY, M.D., Physician to Newland's House and Otto House Private Asylums; 21, New Cavendish street.
- 1883 SUTTON, JOHN BLAND, Assistant Surgeon to the Middlesex Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 6.

Elected

- 1896 SWAN, CHARLES ROBERT JOHN ATKIN, M.B., B.Ch., 4, Devonport street, Hyde Park.
- 1890 SYERS, HENRY WALTER, M.D., 40, Wimpole street.
- 1886 SYMONDS, CHARTERS JAMES, M.S., Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; 26, Weymouth street, Portland place.
- 1875 TAY, WARREN, Senior Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital; Consulting Surgeon to the North-Eastern Hospital for Children, and to the Hospital for Diseases of the Skin, Blackfriars; 4, Finsbury square.
- 1873 TAYLOR, FREDERICK, M.D., *Trustee*; Physician to, and Lecturer on Medicine at, Guy's Hospital; Consulting Physician to the Evelina Hospital for Sick Children; 20, Wimpole street, Cavendish square. S. 1889-93. C. 1894-5. *Sci. Com.* 1889—. *Referee*, 1887-8. *Trans.* 3.
- 1893 TAYLOR, JAMES, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 49, Welbeck street, Cavendish square. *Trans.* 1.
- 1890 TAYLOR, SEYMOUR, M.D., Assistant Physician, West London Hospital; 16, Seymour street, Portman square.
- 1859 TEGART, EDWARD, 60, Scarsdale Villas, Kensington. C. 1888-9.
- 1874 THIN, GEORGE, M.D., 63, Harley street, Cavendish square. C. 1893-4. *Trans.* 13.
- 1862 THOMPSON, EDMUND SYMES, M.D., Consulting Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Sci. Com.* 1889—. *Referee*, 1876-7. *Trans.* 1.
- 1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; Member of the Société de Chirurgie, Paris; 35, Wimpole street, Cavendish square. V.P. 1888. C. 1869. *Trans.* 8.

Elected

- 1862 **Thompson, REGINALD EDWARD, M.D.**, Consulting Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.
- 1892 **THOMSON, STCLAIR, M.D.**, 28, Queen Anne street, Cavendish square. *Trans.* 1.
- 1892 **THORNE, WILLIAM BEZLY, M.D.**, 53, Upper Brook Street.
- 1876 **THORNTON, JOHN KNOWSLEY, M.B., C.M.**, Consulting Surgeon to the Samaritan Free Hospital for Women and Children; Consulting Surgeon to the Grosvenor Hospital for Women, and to the New Hospital for Women; 49, Montagu square. C. 1891. *Lib. Com.* 1886-90, 1893-95. *Trans.* 5.
- 1889 **TIRARD, NESTOR ISIDORE CHARLES, M.D.**, Professor of Materia Medica and Therapeutics, King's College; Physician to King's College Hospital, and Physician to the Evelina Hospital for Sick Children; 74, Harley street, Cavendish square.
- 1872 **TOMES, CHARLES SISSMORE, M.A., F.R.S.**, 9, Park crescent, Portland place. C. 1887. V.P. 1897—. *Lib. Com.* 1879.
- 1882 **TOOTH, HOWARD HENRY, M.D.**, Assistant Physician, late Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Physician to the Metropolitan Hospital; Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square; 34, Harley street, Cavendish square. *Sci. Com.* 1896—.
- 1879 **TREVES, FREDERICK**, Surgeon to, and Lecturer on Surgery at, the London Hospital; 6, Wimpole street, Cavendish square. C. 1895-6. *Referee*, 1890—. *Sci. Com.* 1889-95. *Trans.* 5.
- 1859 **Truman, EDWIN THOMAS**, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.
- 1897 **TUNNICLIFFE, FRANCIS WHITTAKER, M.D.**, 48, Upper Berkeley street, Portman square.

Elected

- 1889 TURNBULL, GEORGE LINDSAY, M.B., Grove House, 76, Ladbroke grove.
- 1875 TURNER, FRANCIS CHARLEWOOD, M.D., Physician to the London Hospital; Consulting Physician to the North-Eastern Hospital for Children; 15, Finsbury square. C. 1895-7.
- 1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 49, Green street, Park lane.
- 1894 TURNER, PHILIP DYMCK, M.D., 44, Welbeck street.
- 1896 TURNER, WILLIAM ALDREN, M.D., 13, Queen Anne street, Cavendish square.
- 1896 TURNEY, HORACE GEORGE, M.B., 68, Portland place.
- 1891 TWEED, REGINALD, M.D., 55, Upper Brook street, Grosvenor square.
- 1892 TWEEDY, JOHN, Professor of Ophthalmic Medicine and Surgery in University College, Ophthalmic Surgeon to University College Hospital, and to the Royal London Ophthalmic Hospital; 100, Harley Street, Cavendish square.
- 1876 VENN, ALBERT JOHN, M.D., 70A, Grosvenor street, and Hemnal Wood, Chislehurst.
- 1870 VENNING, EDGCOMBE, 30, Cadogan place.
- 1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.
- 1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy, and Senior Physician to the French Hospital and Dispensary, Shaftesbury Avenue; 19A, Hanover square.
- 1891 VOELCKER, ARTHUR FRANCIS, M.D., B.S., Assistant Physician to, Pathologist and Curator of the Museum, and Lecturer on Biology at the Middlesex Hospital; 31, Harley street.
- 1896 WAGGETT, ERNEST, M.B., B.C., 45, Upper Brook street.

Elected

- 1886 WAINWRIGHT, BENJAMIN, M.B., C.M., Assistant Surgeon to the Royal Westminster Ophthalmic Hospital; 47, Weymouth street, Portland place.
- 1884 WAKLEY, THOMAS, jun., 5, Queen's Gate, South Kensington.
- 1896 WALDO, FREDERICK JOSEPH, M.D., 1, Plowden Buildings, Temple.
- 1883 WALLER, AUGUSTUS, M.D., F.R.S., Lecturer on Physiology, St. Mary's Hospital; Weston Lodge, 16, Grove End road, St. John's Wood. *Referee*, 1895—.
- 1888 WALLIS, FREDERICK CHARLES, M.B., B.C., Assistant Surgeon to the Charing Cross Hospital; 26, Welbeck street, Cavendish square.
- 1896 WALSHAM, HUGH, M.B., 114, Harley street.
- 1873 WALSHAM, WILLIAM JOHNSON, C.M., Senior Assistant Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 77, Harley street, Cavendish square. C. 1888-9. *Referee*, 1895—. *Lib. Com.* 1882-5. *Trans.* 7.
- 1886 WARD, ALLAN OGIER, M.D., Lansdowne House, High road, Tottenham.
- 1890 WARD, ARTHUR HENRY, Surgeon to Out-patients, Lock Hospital; 7, Hertford street, Mayfair.
- 1891 WARING, HOLBURN JACOB, M.B., B.S., B.Sc., 9, Upper Wimpole street.
- 1877 WARNER, FRANCIS, M.D., Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital; 5, Prince of Wales terrace, Kensington Palace. *Trans.* 1.
- 1889 WASHBOURN, JOHN WYCHENFORD, M.D., Assistant Physician to, Physician in Charge of Electrical Department, Joint Lecturer on Physiology, and Demonstrator of Bacteriology at, Guy's Hospital; Physician to the London Fever Hospital; 6, Cavendish place. *Trans.* 1.

Elected

- 1894 WATENHOUSE, HERBERT FURNIVALL, C.M., Assistant Surgeon to the Charing Cross Hospital; 81, Wimpole street.
- 1861 Watson, WILLIAM SPENCER, M.B., Surgeon to the Throat Department of the Great Northern Central Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.
- 1879 DE WATTEVILLE, ARMAND, M.D., 30, Welbeck street, Cavendish square.
- 1892 WEAVER, FREDERICK POYNTON, M.D., Cedar Lawn, Hampstead Heath.
- 1891 Weber, FREDERIC PARKES, M.D., Physician to the German Hospital, 19, Harley street, W.
- 1857 WEBER, HERMANN, M.D., Consulting Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. V.P. 1885-6. *Sci. Com.* 1889—. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.
- 1896 WEIR, ARTHUR NESHAM, M.B., 55, St. Charles square, Bayswater.
- 1895 WELLS, SYDNEY RUSSELL, M.D., 24, Somerset street, Portman square.
- 1842 West, CHARLES, M.D., Foreign Associate, Académie de Médecine, Paris; 4, Evelyn Mansions, Carlyle place, Victoria street. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.
- 1877 WEST, SAMUEL, M.D., Assistant Physician to St. Bartholomew's Hospital; Senior Physician to the Royal Free Hospital; 15, Wimpole street, Cavendish square. C. 1894-5. *Lib. Com.* 1892-4. *Trans.* 5.
- 1888 WETHERED, FRANK JOSEPH, M.D., Assistant Physician to the Hospital for Consumption, Brompton; 83, Harley street, Cavendish square. *Trans.* 1.
- 1881 WHARRY, ROBERT, M.D., 6, Gordon square.

Elected

- 1875 WHIPHAM, THOMAS TILLYER, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square. C. 1892-8.
- 1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 144, Sloane street.
- 1881 WHITE, WILLIAM HALE, M.D., Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 65, Harley street, Cavendish square. *Referee*, 1888-97. *Trans.* 4.
- 1890 WHITE-COOPER, W. G. O., M.B., 5, Courtfield road, Gloucester road, S.W.
- 1897 WHITFIELD, ARTHUR, M.D., 12, Upper Berkeley street.
- 1877 WHITMORE, WILLIAM TICKLE, Senior Surgeon to the Westminster General Dispensary, to the St. George's and St. James's Dispensary, and to the Gordon Hospital for Diseases of the Rectum; 7, Arlington street, Piccadilly.
- 1863 WILKS, SIR SAMUEL, Bart., M.D., LL.D., F.R.S., Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; Consulting Physician to Guy's Hospital, and Member of the Senate of the University of London; 72, Grosvenor street. *Referee*, 1872-81.
- 1890 WILLCOCKS, FREDERICK, M.D., Physician to Out-Patients, and Lecturer on Materia Medica and Therapeutica, at the Charing Cross Hospital; Physician to the Evelina Hospital for Sick Children; 14, Mandeville place, Manchester square.
- 1865 WILLETT, ALFRED, *Trustee*; Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. V.P. 1890-91. *Referee*, 1882-89, 1892—. *Bldg. Com.* 1889-92. *Ho. Com.* 1892—. *Trans.* 2.
- 1887 WILLETT, EDGAR, M.B., 25, Welbeck street, Cavendish square.
- 1896 WILLIAMS, ALFRED HENRY, M.D., Harrow-on-the-Hill.

Elected

- 1888 WILLIAMS, CAMPBELL, 24, Welbeck street, Cavendish square.
- 1866 WILLIAMS, CHARLES THEODORE, M.D., *Trustee for Debenture-holders*; Consulting Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 2, Upper Brook street, Grosvenor square. C. 1884-5. *Referee*, 1888—. *Lib. Com.* 1880-3. *Sci. Com.* 1889—. *Trans.* 5.
- 1881 WILLIAMS, DAWSON, M.D., Physician to the East London Hospital for Children; 101, Harley street. *Trans.* 1.
- 1872 WILLIAMS, SIR JOHN, Bart., M.D., Physician-Accoucheur to H.R.H. the Princess Beatrice; Emeritus Professor of Obstetric Medicine, University College, London; Consulting Obstetric Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1891. *Referee*, 1878-90. *Lib. Com.* 1876-82.
- 1890 WILLS, WILLIAM ALFRED, M.D., Assistant Physician to the Westminster Hospital; Senior Physician to the North-Eastern Hospital for Children; 29, Lower Seymour street, Portman square.
- 1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.
- 1885 WOLFENDEN, RICHARD NORRIS, M.D., 19, Harley street, Cavendish square.
- 1887 WOOD, THOMAS OUTTERSON, M.D., Senior Physician to the West End Hospital for Nervous Diseases; 40, Margaret street, Cavendish square.
- 1891 WOODFORDE, ALFRED POWNALL, 160, Goldhawk road.
- 1892 WOODHEAD, GERMAN SIMS, M.D., Director of the Research Laboratory, Conjoint Board of R.C.P.Lond. and R.C.S.Eng.; 1, Nightingale lane, Balham.
- 1890 WYNTER, WALTER ESSEX, M.D., Assistant Physician to the Middlesex Hospital; 30, Upper Berkeley street, Portman square.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION

- | | | | |
|------|-------------------------------------|------|-------------------------------------|
| 1838 | Henry Spencer Smith. | 1859 | Wm. Howship Dickinson, M.D. |
| 1840 | Sir James Paget, Bt., F.R.S. | | Edwin Thomas Truman. |
| 1842 | Sir John Simon, K.C.B., F.R.S. | | Richard Barwell. |
| | Charles West, M.D. | | Edward Tegart. |
| 1843 | Henry Lee. | 1860 | William Ogle, M.D. |
| 1845 | Sir Edwin Saunders. | | Thomas Bryant. |
| | Edward U. Berry. | | John Couper. |
| 1848 | Sir Edward H. Sieveking, M.D. | | Henry Howard Hayward. |
| | John Clarke, M.D. | 1861 | William Spencer Watson. |
| 1849 | C. H. F. Routh, M.D. | 1862 | Lionel Smith Beale, M.B., F.R.S. |
| 1850 | Sir R. Quain, Bt., M.D., F.R.S. | | Edmund Symes Thompson, M.D. |
| 1851 | John Birkett. | | Reginald Edward Thompson, M.D. |
| | John A. Kingdon. | | George Cowell. |
| | Bernard E. Brodhurst. | 1863 | Sir Samuel Wilks, Bt., M.D., F.R.S. |
| | Robert J. Spitta, M.D. | | Samuel Fenwick, M.D. |
| 1852 | William Adams. | | Julius Althaus, M.D. |
| | Sir Henry Thompson. | | Sydney Ringer, M.D., F.R.S. |
| 1853 | Robert Brudenell Carter. | | Sir Thomas Smith, Bart. |
| 1854 | Sir Alfred B. Garrod, M.D., F.R.S. | | Arthur B. R. Myers. |
| 1855 | William Marcet, M.D., F.R.S. | | William Sedgwick. |
| 1856 | Charles J. Hare, M.D. | 1864 | John Harley, M.D. |
| | William Bird. | | Thomas William Nunn. |
| | Jonathan Hutchinson, F.R.S. | 1865 | James Edward Pollock, M.D. |
| | Timothy Holmes. | | Reginald Southey, M.D. |
| | Alonzo H. Stocker, M.D. | | George Fielding Blandford, M.D. |
| 1857 | Sir William Overend Priestley, M.D. | | Sir Dyce Duckworth, M.D. |
| | Hermann Weber, M.D. | | Frederick W. Pavy, M.D., F.R.S. |
| | Henry Cooper Rose, M.D. | | John Langton. |
| | Henry Walter Kiallmark. | | Frederick James Gant. |
| 1858 | John William Ogle, M.D. | | Alfred Willett. |

- 1865 Bowater John Vernon.
Alfred Cooper.
Christopher Heath.
- 1866 Thomas Fitz-Patrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
William Selby Church, M.D.
- 1867 Achille Vintras, M.D.
Sir R. Douglas-Powell, Bart., M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac, Bart.
Thomas Pickering Pick.
- 1868 H. Charlton Bastian, M.D., F.R.S.
Sir W. H. Broadbent, Bart., M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
T. Henry Green, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
- 1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
Thomas Laurence Read.
- 1870 J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
Reginald Harrison.
Robert Leamou Bowles, M.D.
- 1871 William Cayley, M.D.
T. Lauder Brunton, M.D., F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Henry Sutherland, M.D.
George Vivian Poore, M.D.
Benjamin Duke, M.D.
- 1872 T. Gilbert-Smith, M.D.
George B. Brodie, M.D.
Sir John Williams, Bart., M.D.
Sir J. Fayer, M.D., F.R.S.
Charles S. Tones, M.A., F.R.S.
Sir William Bartlett Dalby.
- 1873 William Miller Ord, M.D.
Frederick Taylor, M.D.
Norman Moore, M.D.
John Curnow, M.D.
Sir William R. Gowers, M.D., F.R.S.
Sir Wm. Guyer Hunter, M.D.
Jeremiah McCarthy.
Wm. Johnson Smith.
Robert William Parker.
Alex. O. MacKellar.
Henry T. Butlin.
- 1873 Charles Higgens.
William J. Walsham.
- 1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
John Mitchell Bruce, M.D.
Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Henry G. Howse, M.S.
Herbert William Page.
Frederic Durham.
William Robert Smith, M.D.
- 1875 Thomas T. Whipham, M.D.
Francis Charlewood Turner, M.D.
Thomas Crawford Hayes, M.D.
Charles Henry Carter, M.D.
Waren Tay.
Edmund J. Spitta.
Samuel C. Osborn.
Fletcher Beach, M.B.
- 1876 Thomas Barlow, M.D.
Wm. Lewis Dudley, M.D.
Albert J. Venn, M.D.
John Knowsley Thornton, M.B.
N. Charles Macnamara.
John N. C. Davies-Colley, M.C.
- 1877 Sir Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.D.
Alfred Pearce Gould, M.S.
Rickman J. Godlee, M.S.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.D.
John Abercrombie, M.D.
George Allan Heron, M.D.
Joseph A. Ormerod, M.D.
P. Henry Pye-Smith, M.D., F.R.S.
Edward Nettleship.
William Henry Bennett.
William T. Whitmore.
- 1878 Sir Jas. Crichton-Browne, M.D.
Fred. T. Roberts, M.D.
Lord Lister, F.R.S.
Clinton T. Dent.
John H. Morgan.
Donald W. Charles Hood, M.D.
- 1879 Edward Woakes, M.D.
Armand de Watteville, M.D.
Malcolm A. Morris.
A. E. Cumberbatch.
Edmund Owen.
Arthur E. J. Barker.

- 1879 Frederick Treves.
 Horatio Donkin, M.D.
 Thomas John MacLagan, M.D.
 Andrew Clark.
 Francis Henry Champneys, M.D.
 William Watson Cheyne, F.R.S.
 George Henry Savage, M.D.
 H. H. Clutton, M.A.
 Frederic S. Eve.
 E. Noble Smith.
 William Henry Allechin, M.D.
 F. G. Dawtrey Drewitt, M.D.
- 1880 Robert Alex. Gibbons, M.D.
 David Ferrier, M.D., F.R.S.
 Vincent Dormer Harris, M.D.
 Edmund Distin Maddick.
 Jas. John MacWhirter Dunbar, M.D.
 James William Browne, M.B.
 William Appleton Meredith, M.B.
 Malcolm Macdonald McHardy.
 A. Boyce Barrow.
 William Murrell, M.D.
 Leslie Ogilvie, M.B.
 George Ogilvie, M.B.
 Charles Edward Beevor, M.D.
 Thomas Colcott Fox, M.B.
 George Henry Makins.
- 1881 Francis de Havilland Hall, M.D.
 Robert Wharry, M.D.
 Cecil Yates Biss, M.D.
 Richard Clement Lucas, B.S.
 Stephen Mackenzie, M.D.
 William Hale White, M.D.
 Eustace Smith, M.D.
 Percy Kidd, M.D.
 Oswald A. Browne.
 W. Bruce Clarke, M.B.
 Dawson Williams, M.D.
 George Lindsay Johnson, M.D.
 Henry Edward Juler.
 Jonathan F. C. H. Macready.
 C. B. Lockwood.
- 1882 Philip J. Hensley, M.D.
 Ernest Clarke, M.D.
 John Barclay Scriven.
 George Robertson Turner.
 Howard Henry Tooth, M.D.
 Herbert Isambard Owen, M.D.
 Charles R. B. Keetley.
 Anthony A. Bowlby.
 Amand J. McC. Routh, M.D.
 Seymour J. Sharkey, M.D.
 William Lang.
 Henry Radcliffe Crocker, M.D.
- 1883 Edwin Clifford Beale, M.A., M.B.
 James Kingston Fowler, M.D.
 James Frederic Goodhart, M.D.
 John Charles Galton, M.A.
 W. Hamilton A. Jacobson, M.Ch.
 Walter H. Jessop, M.B.
 Walter Edmunds, M.C.
 Victor A. Horsley, F.R.S.
 Dudley Wilmot Buxton, M.D.
 Charles Douglas F. Phillips, M.D.
 John James Pringle, M.B.
 Henry Roxburgh Fuller, M.D.
 Wilmot Parker Herringham, M.D.
 Augustus Waller, M.D.
 William Pasteur, M.D.
 Edward Albert Schäfer, F.R.S.
 John Bland Sutton.
 William Rose, M.B.
 Storer Bennett.
 Robert Marcus Gunn, M.B.
 James Dixon Bradshaw, M.B.
- 1884 George Newton Pitt, M.D.
 Charles Stonham.
 Stanley Boyd, M.B.
 William Arbuthnot Lane, M.S.
 Arthur Marmaduke Sheild, M.B.
 Frederic Bowreman Jessett.
 Sidney Harris Cox Martin, M.D.
 George Lawson.
 Thomas Wakley, jun.
 F. Swinford Edwards.
 James Johnston, M.D.
 William Duncan, M.D.
 Charles Chinner Fuller.
 Jean Samuel Keser, M.D.
 George Richard Turner Phillips.
 Bilton Pollard.
- 1885 Alexander Haig, M.D.
 Theodore Dyke Acland, M.D.
 Frederick Walker Mott, M.D.
 James Berry.
 John Cahill, M.D.
 John Poland.
 Heinrich Port, M.D.
 R. Norris Wolfenden, M.D.
 A. C. Butler-Smythe.
 Charles Alfred Ballance, M.S.
 Walter S. A. Griffith, M.D.
 John Edward Squire, M.D.
 John D. Malcolm, M.B., C.M.
 Phineas S. Abraham, M.D.
 Henry Willingham Gell, M.B.
- 1886 Robert Maguire, M.D.
 Harrington Sainsbury, M.D.

- 1886 Cuthbert Hilton Golding-Bird, M.B.
Benjamin Wainewright, M.B., C.M.
Lauriston Elgie Shaw, M.D.
Charters James Symonds, M.S.
Robert Boxall, M.D.
Allan Ogier Ward, M.D.
Archibald Edward Garrod, M.D.
Stephen Paget.
William Radford Dakin, M.D.
Samuel Herbert Habershon, M.D.
Arthur Quarry Silcock.
Arthur H. N. Lewers, M.D.
- 1887 Walter George Spencer.
Thomas Outterson Wood, M.D.
Edgar William Willett, M.B.
Henry Lewis Jones, M.D.
Francis George Penrose, M.D.
Hugh Percy Dunn.
Frederic William Hewitt, M.D.
Harry Scott, M.D.
James Barry Ball, M.D.
Gilbert Richardson, M.D.
D'Arcy Power, M.B.
Charles Arkle, M.D.
John Gay.
James Calvert, M.D.
Percy J. F. Lush, M.B.
- 1888 Robert Henry Scanes Spicer, M.D.
Jonathan Hutchinson, jun.
Campbell Williams.
James Donelan, M.B., C.M.
John Anderson, M.D., C.I.E.
Laurie Asher Lawrence.
Arthur Pearson Luff, M.D., B.Sc.
Albert Carless, M.B., B.S.
Frederick C. Wallis, M.B., B.C.
Charles James Cullingworth, M.D.
Edmund Cautley, M.D., B.C.
H. Montague Murray, M.D.
Arthur Symons Eccles, M.B.
Frank Joseph Wethered, M.D.
Edmund Wilkinson Roughton, M.D.
Frederick William Cock, M.D.
John Phillips, M.D.
- 1889 Montagu Handfield-Jones, M.D.
Norman M. MacLehose, M.B.
David Henry Goodsall.
Raymond Johnson, M.B.
John Fletcher Little, M.B.
Henry Work Dodd.
George Lindsay Turnbull, M.B.
Sir William Roberts, M.D., F.R.S.
Sidney Phillips, M.D.
William Charles Bull, M.B.
- 1889 George P. Field.
John Wychenford Washbourn, M.D.
Charles Henry Cosens.
Henry Percy Dean, M.B., M.S.
Alfred Samuel Gubb.
William Hunter, M.D.
J. Inglis Parsons, M.D.
Bernard Pitts, M.B., M.C.
Robert Percy Smith, M.D., B.S.
Herbert R. Spencer, M.D., B.S.
Nestor Isidore Chas. Tirard, M.D.
John Rose Bradford, M.D., F.R.S.
Roland Danvers Brinton, M.D.
James Cagney, M.D.
Charles D. B. Hale, M.D.
Edwin Cooper Perry, M.D.
Morton Smale.
Frederick Willcocks, M.D.
R. Ashton Postock.
William T. Holmes Spicer, M.B.
Thomas Henry Crowle.
Henry Walter Syers, M.D.
Seymour Taylor, M.D.
William Alfred Wills, M.D.
G. O. White-Cooper, M.B.
Herbert William Allingham.
William Anderson.
William A. F. Bateman.
James Jackson Clarke, M.B.
Leonard G. Guthrie, M.B., B.Ch.
G. William Hill, M.D., B.Sc.
Edward Law, M.D., C.M.
Patrick Manson, M.D., C.M.
Humphry D. Rolleston, M.D., B.C.
Arthur Henry Ward.
Walter Essex Wynter, M.D., B.S.
- 1891 William Lee Dickinson, M.D.
Herbert P. Hawkins, M.D., B.Ch.
Cyril Ogle, M.A., M.B.
Leonard Remfry, M.D.
Arthur F. Voelcker, M.D., B.S.
Alfred Pownall Woodforde.
Herbert T. Herring, M.B., B.S.
Ernest Muirhead Little.
Henry Charrington Martin, M.D.
Frederick William Andrewes, M.B.
Alfred Eddowes, M.D.
Herbert Morley Fletcher, M.D.
William Heaton Hamer, M.D.
William Bromfield Paterson.
Reginald Tweed, M.D.
Holburt Jacob Waring.
Frederic Parkes Weber, M.D.
F. E. Batten, M.D.

- 1891 Thomas Jessopp Bokenham.
 Norman Dalton, M.D.
 P. R. W. De Santi.
 P. W. Dove.
 William J. Gow, M.D.
 Charles Arthur Mercier, M.B.
 Paul Frank Moline, M.B.
 Edward Percy Paton, M.D.
 Arthur Bowen Rendel, M.B., B.C.
 James Samuel Risien Russell, M.B.
 George Cockburn Smith.
 Charles Percival White, M.B., B.C.
 W. Page May, M.D.
 Richard J. Reece.
- 1892 Edward Cotterell.
 J. Dundas Grant, M.D.
 R. J. Bliss Howard, M.D.
 Thomas Horrocks Openshaw, M.B.
 William Bezly Thorne, M.D.
 German Sims Woodhead, M.D.
 W. H. Russell Forsbrook, M.D.
 John Harold.
 William Ward Leadam, M.D.
 John Alfred Masters, M.D.
 Gustave Schorstein, M.B.
 Charles Sempill de Segundo.
 John Tweedy.
 E. H. Myddelton-Gavey.
 E. Matthews James.
 J. S. Selwyn-Harvey, M.D.
 StClair Thomson, M.D.
 F. Manley B. Sims.
 Solomon Charles Smith, M.D.
 F. Poynton Weaver, M.D.
 Henry Rayner, M.D.
- 1893 James Taylor, M.D.
 Howard Barrett.
 Robert Cozens Bailey, M.B.
 Henry Albert Caley, M.D.
 Arthur Edward Giles, M.D.
 Miles Miley, M.B.
 Alfred A. Kanthack, M.D.
 Kenneth McLeod, M.D.
 D. Watkin Roberts, M.D.
 Leonard A. Bidwell.
 Frédéric F. Burghard, M.D., M.S.
 William McAdam Eccles, M.S.
 Vaughan Harley, M.D.
 George Herschell, M.D.
 Arnold Lawson.
 Guthrie Rankin.
 Walter Knowsley Sibley, M.D.
 Richard Sisley, M.D.
- 1894 Richard Gill.
- 1894 Joseph Sefton Sewill.
 Thomas Vincent Dickinson, M.D.
 Herbert Edward Durham, M.B.
 Alexander Morison, M.D.
 L. Hemington Pegler, M.D.
 Herbt. Furnivall Waterhouse, C.M.
 Philip D. Turner, M.D.
 Percy Furnivall.
 R. L. Langdon-Down, M.B., B.C.
 Allan Macfadyen, M.D., B.S.
 Ernst Michels, M.D.
 Wm. Rivers Pollock, M.B., B.C.
 Charles Slater, M.B.
- 1895 Charles Arthur Parker.
 Sydney Russell Wells, M.D.
 Alfred Milne Gossage, M.B.
 Robert Murray Leslie, M.B.
 Gerald R. Baldwin.
 James Galloway, M.D.
 David Bridge Lees, M.D.
 Arthur G. Phear, M.D.
- 1896 Joseph Lockhart Downes, M.B.
 Edward Wilberforce Goodall, M.D.
 James Ernest Lane.
 George Oliver, M.D.
 George Alex. Sutherland, M.D.
 Charles F. Buttar, M.B.
 P. J. Freyer, M.D., I.M.S., M.A.
 Percival Horton-Smith, M.B.
 Frederick Henry Lewis, M.B.
 James Keogh Murphy, M.B.
 Thomas William Shore, M.D.
 John Stretton Sloane, M.B.
 William Aldren Turner, M.D.
 Arthur Nesham Weir, M.B.
 John Brian Christopherson, M.B.
 Charles Hubert Roberts, M.D.
 John W. Watson Stephens, M.B.
 Charles R. J. Atkin Swan, M.B.
 James Kingston Barton.
 J. Walter Carr, M.D.
 John H. Dauber, M.A., M.B., B.Ch.
 Alexander Grant Russell Foulerton.
 L. Vernon Jones, B.A., M.D., B.Ch.
 Alexander MacGregor, M.D.
 Henry Betham Robinson.
 Horace George Turney, M.B.
 Ernest Waggett, M.B., B.C.
 Frederick Joseph Waldo, M.B.
 Hugh Walsham, M.B.
- 1897 Comyns Berkeley, M.B., B.C.
 William Arthur Brailey, M.D.
 James Cantlie, M.B.
 Raymond H. Payne Crawford, M.D.

1897	Louis Jenner, M.B. W. J. Collings Merry, M.D., B.Ch. Charles Herbert Perram, M.D. Francis Whittaker Tunncliffe, M.D.	1897	Arthur Whitfield, M.D. Arthur A. Jamison, M.D. Edward Stainer, M.A., M.B.
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The following Non-resident Fellows pay a subscription of £3 3s., and are thereby entitled to all the privileges of Resident Fellows.

Elected

- 1891 BRODIE, CHARLES GORDON, Fernhill, Wootton Bridge, Isle of Wight.
- 1888 CLARKE, ROBERT HENRY, M.B., Westwood, Isle of Thanet, Kent.
- 1884 DRAGE, LOVELL, M.A., M.B., B.Ch.Oxon., Burleigh Mead, Hatfield, Herts.
- 1886 LEDIARD, HENRY AMBROSE, M.D., Surgeon to the Cumberland Infirmary ; 35, Lowther street, Carlisle.
- 1887 PAGET, CHARLES EDWARD, Medical Officer of Health for the County Borough of Salford ; Lecturer on Public Health, Owen's College, Victoria University ; North Bentcliffe, Eccles, Lancashire.
- 1882 REID, THOMAS WHITEHEAD, M.D., Surgeon to the Kent and Canterbury Hospital ; St. George's House, Canterbury.
- 1891 RUFFER, MARC ARMAND, Medical School, Cairo.
- 1887 WALLACE, EDWARD JAMES, M.D., Holmbush, Grove road, Southsea.

NON-RESIDENT FELLOWS

Elected

- 1851 *Acland, SIR HENRY W.*, Bart., K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; late Regius Professor of Medicine in the University of Oxford; Oxford.
- 1839 *ALCOCK, SIR RUTHERFORD*, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Peking. *Trans.* 1.
- 1866 *ALLBUTT, THOMAS CLIFFORD*, M.D., LL.D. Glasgow, F.R.S., Regius Professor of Physic, Univ. Camb.; Consulting Physician to the Leeds General Infirmary; St. Rhadegund's, Cambridge. *Trans.* 8.
- 1884 *ANDERSON, ALEXANDER RICHARD*, Surgeon to the General Hospital, 5, East Circus Street, Nottingham. *Trans.* 1.
- 1880 *Appleton, HENRY*, M.D., 2, Swinburn villas, Romford, Essex.
- 1873 *Baker, J. WRIGHT*, Consulting Surgeon to the Derbyshire General Infirmary [care of Dr. Benthall, 101, Friar gate, Derby].
- 1896 *BAGSHAW, FREDERIC*, M.D., 35, Warrior Square, St. Leonard's-on-Sea.
- 1891 *BALGARNIE, WILFRED*, M.B., The Dutch House, Hartley Wintney, Winchfield.
- 1896 *BALL, CHARLES BENT*, M.D., Ch.M., 24, Merrion square North, Dublin.

Elected

- 1866 *Banks, SIR JOHN*, M.D., LL.D., D.Sc., K.C.B., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to Sir Patrick Dun's and City of Dublin Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Royal University in Ireland; 45, Merrion square, Dublin.
- 1886 *BANKS, WILLIAM MITCHELL*, M.D., Surgeon to the Liverpool Royal Infirmary; 28, Rodney street, Liverpool.
- 1882 *BARKER, FREDERICK CHARLES*, M.D., Surgeon-Major, Bombay Medical Service.
- 1881 *Barnes, HENRY*, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.
- 1861 *Barnes, ROBERT*, M.D., Liss, Hants. C. 1877-8. V.P. 1889-90. *Referee*, 1867-76, 1891—. *Lib. Com.* 1869-73. *Sci. Com.* 1889—. *Trans.* 4.
- 1840 *BARROW, BENJAMIN*, Consulting Surgeon to the Royal Isle of Wight Infirmary; Southlands Park road, Ryde.
- 1860 *Bealey, ADAM*, M.D., M.A., Filsham Lodge, Filsham road, St. Leonard's-on-Sea, Sussex.
- 1856 *BEARDSLEY, AMOS*, F.L.S., The Towers, Grange-over-Sands, Lancashire.
- 1896 *BELBEN, FRANK*, M.B., Hoo Meavy, Branksome Chine, Bournemouth.
- 1880 *BENNETT, ALEXANDER HUGHES*, M.D. (Travelling).
- 1889 *BENTLEY, ARTHUR J. M.*, M.D., Mena House, Pyramids, Cairo, Egypt.
- 1872 *BEVERLEY, MICHAEL*, M.D., Surgeon to the Norfolk and Norwich Hospital; 54, Prince of Wales road, Norwich.
- 1865 *Bickersteth, EDWARD ROBERT*, Consulting Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool. *Trans.* 1.
- 1892 *BICKERSTETH, ROBERT ALEXANDER*, M.A., M.B., Assistant Surgeon to the Liverpool Royal Infirmary; 2, Rodney street, Liverpool.

Elected

- 1878 *BINDON, WILLIAM JOHN VERREKER*, M.D., 48, St. Ann's street, Manchester.
- 1849 *Birkett, EDMUND LLOYD*, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; Westbourne Rectory, Emsworth, Hampshire. C. 1865-6. *Referee*, 1851-9.
- 1865 *BLANCHET, HILARION*, Examiner to the College of Physicians and Surgeons, Lower Canada; 35, Conillard street, Quebec, Canada.
- 1869 *BOURNE, WALTER*, M.D. (Travelling).
- 1874 *BRADSHAW, A. F.*, C.B., Surgeon Major-General, Principal Medical Officer, H.M.'s Forces in India; Simla, India. [Agents: Holt & Co., 17, Whitehall place.]
- 1876 *BRIDGES, ROBERT*, M.B., Manor House, Yattendon, Newbury, Berks.
- 1867 *BRIDGEWATER, THOMAS*, M.B., LL.D., Harrow-on-the-Hill, Middlesex.
- 1891 *BRODIE, CHARLES GORDON*, Fernhill, Wootton Bridge, Isle of Wight.
- 1892 *BRONNER, ADOLPH*, M.D., 33, Manor row, Bradford.
- 1894 *BROOK, WILLIAM HENRY BREFFIT*, 1, James street, Lincoln.
- 1888 *BROWNE, HENRY LANGLEY*, Moor House, West Bromwich.
- 1881 *BROWNE, JOHN WALTON*, M.D., Surgeon to the Belfast Royal Hospital; Surgeon to the Belfast Ophthalmic Hospital; 10, College square N., Belfast.
- 1864 *BUCKLE, FLEETWOOD*, M.D., Merton Lodge, Merton road, Southsea.
- 1851 *Cadge, WILLIAM*, Consulting Surgeon to the Norfolk and Norwich Hospital; 49, St. Giles's street, Norwich. *Trans.* 1.
- 1891 *CAMPBELL, HENRY JOHNSTONE*, M.D., 157, Manningham lane, Bradford.

Elected

- 1888 *CARTER, WILLIAM JEFFREYS BECHER*, Aliwal North, Cape Colony.
- 1884 *CHAFFEY, WAYLAND CHARLES*, M.D., Physician to the Royal Alexandra Hospital for Children; 13, Montpellier road, Brighton.
- 1859 *CHANCE, FRANK*, M.B., Burleigh House, Sydenham hill.
- 1885 *CHAPMAN, PAUL MORGAN*, M.D., Physician to the Hereford General Infirmary, 1, St. John street, Hereford. *Trans.* 1.
- 1881 *Chavasse, THOMAS FREDERICK*, M.D., C.M., Surgeon to the Birmingham General Hospital; Consulting Surgeon to the Bromsgrove Hospital; 22, Temple row, Birmingham. *Trans.* 3.
- 1873 *Chisholm, EDWIN*, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.
- 1892 *CLARK, JAMES CHARLES*, 35, Castle road, Bedford.
- 1888 *CLARKE, ROBERT HENRY*, M.B., Westwood, Ramsgate.
- 1857 *COATES, CHARLES*, M.D., Consulting Physician to the Bath Royal United Hospital; 10, Circus, Bath.
- 1868 *COCKLE, JOHN*, A.M., M.D., F.L.S., Consulting Physician to the Royal Free Hospital; The Lodge, West Molesey. *Trans.* 2.
- 1893 *COLB, ROBERT HENRY*, M.D., Moorcroft, Hillingdon, Uxbridge.
- 1891 *COOK, HERBERT GEORGE*, M.D., B.S., 22, Newport road, Cardiff.
- 1891 *COUNBE, JOHN BATTEN*, M.D., Rosslyn, Clevedon, Somerset.
- 1869 *Cresswell, PEARSON B.*, Surgeon to the Merthyr General Hospital; Dowlais, Merthyr Tydvil.
- 1892 *CROSS, FRANCIS RICHARDSON*, M.B., Ophthalmic Surgeon to the Bristol Royal Infirmary, and Surgeon to the Bristol Eye Hospital; Worcester House, Clifton, Bristol.

Elected

- 1895 *DARDEL, JEAN*, M.D., Aix-les-Bains, Savoy.
- 1879 *DARWIN, FRANCIS*, M.B., F.R.S., Wychfield, Huntingdon road, Cambridge.
- 1874 *DAVIDSON, ALEXANDER*, M.D., Physician to the Liverpool Royal Infirmary; 2, Gambier terrace, Liverpool.
- 1878 *DAVY, RICHARD*, Consulting Surgeon to the Westminster Hospital; Burstone House, Bow, North Devon. *Trans.* 1.
- 1882 *DAWSON, YELVERTON*, M.D., Heathlands, Southbourne-on-Sea, Hants.
- 1889 *DELEPINE, SHERIDAN*, B.S., M.B., Professor of Pathology, Owens College, Manchester. *Trans.* 1.
- 1867 *DRAGE, CHARLES*, M.D., Hatfield, Herts.
- 1884 *DRAGE, LOVELL*, M.D., B.Ch. Oxon., Burleigh Mead, Hatfield, Herts.
- 1885 *DRUMMOND, DAVID*, M.D., 7, Saville place, Newcastle-on-Tyne.
- 1880 *DRURY, CHARLES DENNIS HILL*, M.D., Bondgate, Darlington.
- 1871 *DUKES, CLEMENT*, M.D., B.S., Physician to Rugby School, and Senior Physician to the Hospital of St. Cross, Rugby; Sunnyside, Rugby, Warwickshire.
- 1867 *DUKES, MAJOR CHARLES*, M.D., Clarence Villa, Torrapark, Ilfracombe.
- 1889 *DUNCAN, JOHN*, M.D., St. Petersburg, Russia.
- 1843 *DUREANT, CHRISTOPHER MERCEER*, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.
- 1872 *EAGER, REGINALD*, M.D., Northwoods, near Bristol.
- 1887 *EASMON, JOHN FARRELL*, M.D., Assistant Colonial Surgeon, Gold Coast Colony, and Acting Chief Medical Officer of the Colony; Accra, Gold Coast, West Africa.

Elected

- 1887 *ELLIOTT, JOHN*, Whitefriars Lodge, Chester.
- 1848 *ELLIS, GEORGE VINER*, Minsterworth, Gloucester. C.
1863-4. *Trans.* 2.
- 1868 *ELLIS, JAMES, M.D.*, The Sanatorium, Anaheim, Los
Angeles County, California.
- 1889 *ELLISTON, WILLIAM ALFRED, M.D.*, Stoke Hall, Ipswich.
- 1875 *Fagan, JOHN*, Surgeon to, and Lecturer on Clinical Sur-
gery at, the Belfast Royal Hospital; 19, Great Victoria
street, Belfast.
- 1897 *FAGGE, THOMAS HENRI, M.D.*, Villa de la Porte Rouge,
Monte Carlo.
- 1869 *FAIRBANK, FREDERICK ROYSTON, M.D.*, Hillside, Westcott,
Dorking.
- 1872 *Fenwick, JOHN C. J., M.D.*, Physician to the Durham
County Hospital; Long Framlington, Morpeth.
- 1879 *FINLAY, DAVID WHITE, M.D.*, Professor of the Practice
of Medicine in the University of Aberdeen; Physi-
cian to the Aberdeen Royal Infirmary; Consulting
Physician to the Royal Hospital for Diseases of the
Chest, London; 2, Queen's terrace, Aberdeen. *Referee*,
1891-3. *Trans.* 2.
- 1842 *FLETCHER, THOMAS BELL ELCOCK, M.D.*, Consulting Physi-
cian to the Birmingham General Hospital; 8, Clarendon
crescent, Leamington. *Trans.* 1.
- 1864 *Folker, WILLIAM HENRY*, Consulting and late Hon.
Surgeon to the North Staffordshire Infirmary; Bedford
House, Hanley, Staffordshire.
- 1896 *FORESTIER, HENRI, M.D.*, Aix-les-Bains, Savoie, France.
- 1892 *FOSTER, MICHAEL GEORGE, M.A., M.B.*, Villa Annita,
San Remo.
- 1859 *FOX, EDWARD LONG, M.D.*, Consulting Physician to the
Bristol Royal Infirmary; Church House, Clifton,
Gloucestershire.
- 1871 *FRANK, PHILIP, M.D.*, Cannes, France.

Elected

- 1884 *Franks, KENDAL*, M.D., Kimberley, S. Africa. *Trans.* 2.
- 1889 *Freeman, HENRY WILLIAM*, 24, The Circus, Bath.
- 1876 *FUENER, WILLOUGHBY*, M.D., Surgeon to the Sussex County Hospital; Brunswick square, Brighton.
- 1864 *Gairdner, WILLIAM TENNANT*, M.D., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow. *Trans.* 1.
- 1885 *GAMGEE, ARTHUR*, M.D., F.R.S., Montreux, Switzerland.
- 1867 *GARLAND, EDWARD CHARLES*, Yeovil, Somerset.
- 1867 *GARLIKE, THOMAS W.*, Malvern Cottage, Churchfield road, Ealing.
- 1879 *GARSTANG, THOMAS WALTER HARROP*, Headingley House, Knutsford, Cheshire.
- 1889 *Gaskell, WALTER HOLBROOK*, M.D., F.R.S., Lecturer on Physiology, University of Cambridge; The Uplands, Great Shelford, Cambs.
- 1884 *GIBBES, HENEAGE*, M.D., Professor of Pathology in the University of Michigan; Ann Arbor, Michigan, U.S.A.
- 1897 *GIBSON, GEORGE ALEXANDER*, M.D., D.Sc., 17, Alva Street, Edinburgh.
- 1897 *GILFORD, HASTINGS*, Norwood House, King's road, Reading.
- 1893 *GORDON, WILLIAM*, M.B., M.C.
- 1890 *Gordon, WILLIAM*, M.D., Barnfield Lodge, Exeter.
- 1889 *GREENE, GEORGE EDWARD JOSEPH*, Monte Vista, Ferns, County Wexford.
- 1875 *Greenfield, WILLIAM SMITH*, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.
- 1882 *GRESSWELL, DAN ASTLEY*, M.D., D.P.H., Melbourne, Victoria.

Elected

- 1889 **GRIFFITHS, JOSEPH**, M.A., M.D., C.M., Assistant to the Professor of Surgery in the University of Cambridge; 63, Trumpington street, Cambridge.
- 1862 **GROVE, JOHN**, Pitt House, 15, Johnstown street, Bath.
- 1870 **HAMILTON, ROBERT**, Consulting Surgeon to the Royal Southern Hospital, Liverpool; Magheraybuoy, Portrush, Co. Antrim, Ireland.
- 1892 **HARSANT, WILLIAM HENRY**, The Tower House, Clifton, Bristol.
- 1854 **HAVILAND, ALFRED**, Douglas, Isle of Man.
- 1890 **HAVILAND, FRANK PAPILLON**, M.D., B.C., 57, Warrior square, St. Leonard's-on-Sea.
- 1885 **HAWKINS, FRANCIS HENRY**, M.B., Physician to the Royal Berkshire Hospital; 26, Portland place, Reading.
- 1861 **HAYWARD, WILLIAM HENRY**, Oxford road, Burnley, Lancashire.
- 1895 **HENDERSON, EDWARD ERSKINE**, B.A., M.B., B.C., Brunsfield Lodge, Bromley, Kent.
- 1843 **Holden, LUTHER**, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.
- 1894 **HOLLAND, JAMES FRANK**, M.D., St. Moritz, Engadine, Switzerland.
- 1868 **HOLLIS, WILLIAM AINSLIE**, M.D., Physician to the Sussex County Hospital; 8, Cambridge road, Brighton. *Trans.* 1.
- 1846 **Holthouse, CARSTEN**, 1, Bath terrace, Richmond. C. 1863. *Referee*, 1870-6. *Lib. Com.* 1859-60.
- 1865 **HOWARD, BENJAMIN**, M.D. [New York, U.S.A.] *Trans.* 1.
- 1881 **HOWARD, HENRY**, M.B., Medical Officer of Health, Williamstown, Melbourne, Victoria.
- 1896 **HUGHES, MATTHEW LOUIS**, Surg.-Capt., A.M.S. [care of Messrs. Holt and Co.]. *Trans.* 1.
- 1882 **HUMPHREY, LAURENCE**, M.D., 3, Trinity street, Cambridge.
- 1849 **HUSSEY, EDWARD LAW**, 24, Winchester road, Oxford. *Trans.* 1.

Elected

- 1896 *HYDE, SAMUEL*, M.D., Lismore House, 3, Hardwick street, Buxton.
- 1847 *IMAGE, WILLIAM EDMUND*, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.
- 1863 *JACKSON, THOMAS VINCENT*, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; Whetstone House, Waterloo road south, Wolverhampton.
- 1883 *Jenkins, EDWARD JOHNSTONE*, M.D., The Australian Club, Sydney, New South Wales.
- 1851 *Jenner, SIR WILLIAM*, Bart., M.D., G.C.B., D.C.L., LL.D.Cantab., LL.D.Edin., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Profesaor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Greenwood, Bishop's Waltham, Hants. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.
- 1881 *JENNINGS, WILLIAM OSCAR*, M.D., 35, Rue Marbœuf, Avenue des Champs-Élysées, Paris.
- 1851 *JOHNSON, EDMUND CHARLES*, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Gènevois."
- 1889 *JOHNSON, HAROLD J.*, Senior Assistant, Gloucester County Asylum, Gloucester.
- 1848 *JOHNSTONE, ATHOL ARCHIBALD WOOD*, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.
- 1876 *JONES, LESLIE HUDSON*, M.D., Limefield House, Cheetham hill, Manchester.
- 1875 *Jones, PHILIP SYDNEY*, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, and Fellow of the Senate, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., Wool Exchange, Coleman Street, E.C.]
- 1865 *JORDAN, FURNEAUX*, Consulting Surgeon to the Queen's Hospital, Birmingham; Selly Hill, Birmingham.

Elected

- 1872 *KELLY, CHARLES*, M.D., Professor of Hygiene in King's College, London; Medical Officer of Health for the West Sussex Combined Sanitary District; Ellesmere, Gratwicke road, Worthing, Sussex.
- 1848 *Kendell, DANIEL BURTON*, M.B., Thornhill House, Walton, near Wakefield, Yorkshire.
- 1890 *Kerr, J. G. DOUGLAS*, M.B., C.M., 6, The Circus, Bath.
- 1877 *Khory, RUSTOMJEE NASERWANJEE*, M.D., Hormazd Villa, Khumballa hill, Bombay.
- 1888 *KYNSEY, SIR WILLIAM RAYMOND*, C.M.G., Inspector-General of Hospitals, Colombo, Ceylon.
- 1889 *LANCASTER, ERNEST LE CRONIER*, M.B., B.Ch., Assistant Physician to the Swansea Hospital; Winchester House, Swansea, S. Wales.
- 1873 *Larcher, O.*, M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.
- 1862 *LATHAM, PETER WALLWORK*, M.D., late Downing Professor of Medicine, Cambridge University; Senior Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.
- 1890 *Lawrie, EDWARD*, M.B., Surgeon Lieutenant-Colonel, Indian Medical Department; Residency Surgeon; Hyderabad, Deccan.
- 1880 *LAYCOCK, GEORGE LOCKWOOD*, M.B., C.M., Melbourne, Victoria, Australia.
- 1892 *LAZARUS-BARLOW, WALTER SYDNEY*, M.B., 17, Chesterton road, Cambridge. *Sci. Com.* 1892—.
- 1886 *Lediard, HENRY AMBROSE*, M.D., Surgeon to the Cumberland Infirmary; 35, Lowther street, Carlisle. *Trans.* 1.
- 1882 *LEDWICH, EDWARD L'ESTRANGE*, Anatomist to the Royal College of Surgeons, Ireland; 30, Upper Fitzwilliam street, Dublin.

Elected

- 1896 *LEE, WILLIAM EDWARD*, M.B., 25, St. Peter's hill, Grantham.
- 1895 *LEECH, DANIEL JOHN*, M.D., Elm House, Whalley Range, Manchester. *Sci. Com.* 1896—.
- 1883 *LEESON, JOHN RUDD*, M.D., C.M., Clifden House, Twickenham.
- 1869 *LEGG, JOHN WICKHAM*, M.D. C. 1886. *Referee*, 1882-5. *Lib. Com.* 1878-85. *Trans.* 2.
- 1872 *Little, DAVID*, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; Ophthalmic Surgeon to the Manchester Royal Infirmary; Lecturer on Ophthalmology at the Victoria University; 21, St. John street, Manchester.
- 1889 *Little, JAMES*, M.D., Physician to the Adelaide Hospital; Consulting Physician to the Rotunda, St. Mark's, Steevens', and the Children's Hospitals; 14, Stephen's Green North, Dublin.
- 1871 *LITTLE, LOUIS STROMEYER*, Shanghai, China.
- 1894 *LOWE, THOMAS PAGAN*, 16, The Circus, Bath.
- 1883 *LUND, EDWARD*, Emeritus Professor of Surgery, Victoria University, Manchester; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.
- 1867 *MABERLY, GEORGE FREDERICK*, Mailai Valley, Nelson, New Zealand.
- 1889 *MACALISTER, DONALD*, B.Sc., M.D., Physician to Addenbrooke's Hospital; Lecturer on Medicine, St. John's College; University Lecturer in Medicine; St. John's College, Cambridge.
- 1887 *MACDONALD, GEORGE CHILDS*, M.D.
- 1866 *MACGOWAN, ALEXANDER THORBURN*, M.D.
- 1859 *M'Intyre, JOHN*, M.D., LL.D., Odiham, Hants.
- 1876 *MACKAY, EDWARD*, M.D., Senior Physician to the Royal Alexandra Hospital for Sick Children; Assistant Physician to the Sussex County Hospital; 56, Lansdown place, Brighton.

Elected

- 1854 *Mackinder, DRAPER, M.D.*, Consulting Surgeon to the Dispensary; The Cedars, Gainsborough, Lincolnshire.
- 1891 *MANBY, ALAN REEVE, M.D.*, Surgeon Apothecary to their Royal Highnesses the Prince and Princess of Wales at Sandringham; East Rudham, Norfolk.
- 1894 *MARRIOTT, CHARLES WILLIAM, M.D.*, Aubrey House, Bath road, Reading.
- 1892 *MARTIN, CHRISTOPHER, M.B., C.M.*, 35, George road, Edgbaston, Birmingham.
- 1883 *MAUDSLEY, HENRY CARR, M.D.*, 22, Collins street, Melbourne, Victoria.
- 1839 *MEADE, RICHARD HENRY*, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans. 1.*
- 1895 *MILLS-ROBERTS, ROBERT HERBERT*, Hafod-ty, Llanberis, North Wales.
- 1887 *MIVART, FREDERICK ST. GEORGE, M.D.*, Beaumont Lodge, Worple road, Wimbledon.
- 1896 *MOORE, JOHN WILLIAM, M.D., M.Ch.*, 40, Fitzwilliam square west, Dublin.
- 1891 *MORRIS, GRAHAM*, Wallington, Surrey.
- 1894 *MORSE, THOMAS HERBERT*, 10, Upper Surrey street, Norwich. *Trans. 1.*
- 1873 *MURRAY, J. IVOR, M.D.*, 24, Huntriss row, Scarborough.
- 1881 *NALL, SAMUEL, M.B.*, Dryhurst Lodge, Disley, Stockport.
- 1889 *NAPIER, FRANCIS HORATIO, M.B.*, Cape Town.
- 1870 *NEILD, JAMES EDWARD, M.D.*, Lecturer on Forensic Medicine and Psychological Medicine in the University of Melbourne; 21, Spring street, Melbourne, Victoria.
- 1895 *NEWSHOLME, ARTHUR, M.D.*, 11, Gloucester place, Brighton.
- 1868 *NICHOLLS, JAMES, M.D.*, Trenarren, Newquay, Cornwall.
- 1849 *NORMAN, HENRY BURFORD*, The Manor-house, Drayton, Taunton, Somerset. *Lib. Com. 1857.*
- 1847 *Nourse, WILLIAM EDWARD CHARLES*, Bouverie House, Exeter.
- 1884 *OAKES, ARTHUR, M.D.*, Craycomb, Mount Hermon, Woking.

Elected

- 1880 *O'CONNOR, BERNARD*, A.B., M.D., Physician to the North London Hospital for Consumption; 25, Hamilton road, Ealing.
- 1896 *OGLE, JOHN GILBERT*, M.D., South Redlands, Reigate.
- 1855 *Ogle, WILLIAM*, M.D., late Physician to the Derbyshire Infirmary; The Elms, Duffield road, Derby.
- 1870 *OLDHAM, CHARLES FREDERIC*, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].
- 1883 *Oliver, THOMAS*, M.A., M.D., Professor of Physiology, University of Durham; and Physician to the Newcastle-on-Tyne Infirmary; 7, Ellison place, Newcastle-on-Tyne. *Trans.* 1.
- 1871 *O'Neill, WILLIAM*, M.D., late Physician to the Lincoln Lunatic Hospital, 2, Lindum road, Lincoln.
- 1890 *ORD, WILLIAM WALLIS*, M.D., The Hall, Salisbury.
- 1885 *ORMSBY, L. HEPENSTAL*, M.D., Lecturer on Clinical and Operative Surgery and Surgeon to the Meath Hospital and County Dublin Infirmary; Surgeon to the Children's Hospital, Dublin; 92, Merrion square west, Dublin.
- 1892 *PAGE, HARRY MARMADUKE*, 4, St. Margaret's road, Oxford.
- 1887 *PAGET, CHARLES EDWARD*, Medical Officer of Health for the County Borough of Salford; Lecturer on Public Health, Owens College, Victoria University; North Bentcliffe, Eccles, Lancashire.
- 1858 *Paley, WILLIAM*, M.D., Physician to the Ripon Dispensary; Yore Bank, Ripon, Yorkshire.
- 1887 *PARDINGTON, GEORGE LUCAS*, M.D., 47, Mount Pleasant road, Tunbridge Wells.
- 1885 *PARKER, RUSHTON*, M.B., B.S., Professor of Surgery, University College, Liverpool (Victoria University); Surgeon to the Liverpool Royal Infirmary; 59, Rodney street, Liverpool.
- 1891 *PARKIN, ALFRED*, M.S., M.D., 5, Albion street, Hull. *Trans.* 1.
- 1879 *PEEL, ROBERT*, 120, Collins street east, Melbourne, Victoria.

Elected

- 1874 *PENHALL, JOHN THOMAS*, The Cedars, Broadwas-on-Teme, Worcester.
- 1879 *Pesikaka, HORMASJI DOSABHAI*, 43, Hornby road, Bombay.
- 1878 *Philipson, GEORGE HARE*, M.D., D.C.L., Professor of Medicine in Durham University; Senior Physician to the Newcastle-upon-Tyne Royal Infirmary; 7, Eldon square, Newcastle-upon-Tyne.
- 1891 *PIERCE, BEDFORD*, M.D., The Retreat, York.
- 1841 *Pitman, SIR HENRY ALFRED*, M.D., Consulting Physician to St. George's Hospital; Cranbrook, Bycullah park, Enfield. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.
- 1892 *POWELL, HERBERT ANDREWS*, M.A., M.D., M.Ch., 9, St. Thomas's street, Winchester.
- 1897 *QUARTEY-PAPAFIO, BENJAMIN WILLIAM*, M.B., Accra, Gold Coast.
- 1857 *VON RANKE, HENRY*, M.D., 3, Sophienstrasse, Munich.
- 1890 *RANSOM, WILLIAM BRAMWELL*, M.D., Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.
- 1854 *RANSOM, WILLIAM HENRY*, M.D., F.R.S., Consulting Physician to the Nottingham General Hospital; The Pavement, Nottingham. *Trans.* 1.
- 1882 *REID, SIR JAMES*, M.D., K.C.B., Resident Physician, and Physician in Ordinary to H.M. the Queen, Windsor Castle.
- 1884 *REID, THOMAS WHITEHEAD*, M.D., Surgeon to the Kent and Canterbury Hospital; St. George's House, Canterbury, Kent.
- 1881 *RICE, GEORGE*, M.B., C.M., Sutton, Surrey.
- 1889 *RIVERS, W. H. RIVERS*, M.D., St. John's College, Cambridge.

Elected

- 1871 *Roberts, DAVID LLOYD*, M.D., Obstetric Physician to the Manchester Royal Infirmary; Physician to St. Mary's Hospital, and Lecturer on Clinical Obstetrics and Gynæcology at the Owens College, Manchester; 11, St. John street, Manchester.
- 1889 *ROBERTS, H. LESLIE*, M.B., C.M., 46, Rodney street, Liverpool.
- 1873 *ROBERTSON, WILLIAM HENRY*, M.D., Consulting Physician to the Buxton Bath Charity and Devonshire Hospital; Buxton, Derbyshire.
- 1888 *Robinson, FREDERICK WILLIAM*, M.D., C.M., Huddersfield.
- 1889 *ROBSON, ARTHUR WILLIAM MAYO*, Professor of Surgery, Yorkshire College; Senior Surgeon, Leeds General Infirmary; 7, Park square, Leeds. *Trans.* 4. *Pro.* 1.
- 1885 *ROCKWOOD, WILLIAM GABRIEL*, M.D., Colombo, Ceylon.
- 1850 *ROPER, GEORGE*, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; and to the Royal Infirmary for Children and Women, Waterloo Bridge road; Oulton Lodge, Aylsham, Norfolk. C. 1879-80.
- 1889 *ROSS, DANIEL MCCLURE*, M.D., Cedar Lodge, Littledown Road, Bournemouth.
- 1863 *ROWE, THOMAS SMITH*, M.D., Consulting Surgeon to the Royal Sea-Bathing Infirmary; Union crescent, Margate, Kent.
- 1882 *ROY, CHARLES SMART*, M.D., F.R.S., Professor of Pathology in the University of Cambridge; Trinity College, Cambridge.
- 1871 *RUTHERFORD, WILLIAM*, M.D., F.R.S., Professor of the Institutes of Medicine in the University of Edinburgh; 14, Douglas crescent, Edinburgh.
- 1891 *RUFFEE, MARC ARMAND*, M.D., Medical School, Cairo.
- 1856 *SALTEE, S. JAMES A.*, M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

Elected

- 1855 **SANDERSON, JOHN BURDON**, M.D., LL.D., D.C.L. Durham, D.Sc., F.R.S., Regius Professor of Medicine in the University of Oxford; 64, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.
- 1867 **SANDFORD, FOLLIOTT JAMES**, M.D., Surgeon-Major, Medical Officer of Health of the Drayton Union Rural Sanitary District; Surgeon to the Market Drayton Dispensary; and Consulting Physician to the Market Drayton Cottage Hospital; Market Drayton, Shropshire.
- 1886 **SAUNDBY, ROBERT**, M.D., Physician to the General Hospital, and Consulting Physician to the Hospital for Women, and to the Eye Hospital, Birmingham; Professor of Medicine, Mason College; 83A, Edmund street, Birmingham.
- 1891 **SAUNDERS, FREDERICK WILLIAM**, M.B., B.C., Chieveley House, near Newbury, Berks.
- 1861 **SCOTT, WILLIAM**, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.
- 1897 **SEMPLE, EDWARD**, M.D., Grove house, Fenstanton, Hunts.
- 1887 **SIDEBOTHAM, EDWARD JOHN**, M.B., Erleadene, Bowdon, Cheshire.
- 1857 **SIORDET, JAMES LEWIS**, M.D., Villa Labrolles, Mentone, Alpes Maritimes, France.
- 1886 **SMITH, HOWARD LYON**, Buckland House, Buckland Newton, Dorchester.
- 1894 **SMITH, ROBERT SHINGLETON**, M.D., Deepholm, Clifton Park, Clifton, Bristol.
- 1894 **SMITH, THOMAS RUDOLPH**, M.B., B.C., 25, Bridge road, Stockton-on-Tees.
- 1868 **SOLLY, SAMUEL EDWIN**, Colorado Springs, Colorado, U.S.A.
- 1891 **STEVENS, SURG.-CAPT. CECIL ROBERT**, M.B., B.S., I.M.S., Eden Hospital, Calcutta.

Elected

- 1854 *STEVENS, HENRY*, M.D., late Inspector, Medical Department, Local Government Board, Whitehall; Durham Lodge, St. Margaret's road, Twickenham.
- 1884 *STEWART, EDWARD*, M.D., Brook House, East Grinstead.
- 1879 *Stirling, EDWARD CHARLES*, M.D., Senior Surgeon to the Adelaide Hospital; Lecturer on Physiology in the University of Adelaide, South Australia [care of Messrs. Elder and Co., 7, St. Helen's place].
- 1865 *STOKES, SIR WILLIAM*, M.D., M.C., Surgeon to the Meath Hospital; 5, Merrion square north, Dublin. *Trans.* 1.
- 1871 *STRONG, HENRY JOHN*, M.D., Consulting Surgeon to the Croydon General Hospital; Colonnade House, The Steyne, Worthing.
- 1890 *Sympson, E. MANSEL*, M.D., B.C., Surgeon to the Lincoln County Hospital; Doleraine Court, Lincoln.
- 1870 *TAIT, LAWSON*, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 6.
- 1886 *TEALE, THOMAS PRIDGIN*, M.B., F.R.S., Consulting Surgeon to the Leeds General Infirmary; 38, Cookridge street, Leeds.
- 1890 *THOMAS, WILLIAM ROBERT*, M.D., Little Forest, Bath road, Bournemouth.
- 1891 *THOMSON, JOHN ROBERTS*, M.D., Monkchester, Bournemouth.
- 1883 *THURSFIELD, THOMAS WILLIAM*, M.D., Physician to the Warneford and South Warwickshire General Hospital; Selwood, Beauchamp square, Leamington.
- 1880 *TIVY, WILLIAM JAMES*, 8, Lansdowne place, Clifton, Bristol.
- 1871 *Trend, THEOPHILUS W.*, M.D., Physician to the Royal South Hants Infirmary; 1, Grosvenor square, Southampton.
- 1881 *Treves, WILLIAM KNIGHT*, Surgeon to the National Hospital for Scrofula; 31, Dalby square, Cliftonville, Margate.

Elected

- 1867 *TROTTER, JOHN WILLIAM*, formerly Surgeon-Major, Coldstream Guards ; 4, St. Peter's terrace, York.
- 1873 *TURNER, GEORGE BROWN*, M.D., The Lodge, Hemel Hempsted, Herts.
- 1881 *TYSON, WILLIAM JOSEPH*, M.D., Medical Officer of the Folkestone Infirmary ; 10, Langhorne Gardens, Folkestone.
- 1854 *WADDINGTON, EDWARD*, Hamilton, Auckland, New Zealand.
- 1868 *Walker, ROBERT*, Clovelly, Bideford.
- 1887 *WALLACE, EDWARD JAMES*, M.D., Holmbush, Grove road Southsea.
- 1867 *WALLIS, GEORGE*, Consulting Surgeon to Addenbrooke's Hospital ; 6, Hills road, Cambridge.
- 1883 *Walters, JAMES HOPKINS*, Surgeon to the Royal Berkshire Hospital ; 15, Friar street, Reading.
- 1894 *WARD-HUMPHREYS, GEORGE HERBERT*, Oriel Lodge Cheltenham.
- 1846 *WARR, JAMES THOMAS*, Tilford House, near Farnham, Surrey.
- 1861 *WATERS, A. T. HOUGHTON*, M.D., Consulting Physician to the Royal Infirmary ; 69, Bedford street, Liverpool. *Trans.* 3.
- 1840 *WEBB, WILLIAM WOODHAM*, M.D., Ault-sur-Somme, Somme, France.
- 1874 *WELLS, HARRY*, M.D., San Ysidro, Buenos Ayres, S. America.
- 1882 *WHARRY, CHARLES JOHN*, M.D., 14, Ewell road, Surbiton, Surrey.
- 1897 *WHITE, CHARLES POWELL*, General Hospital, Birmingham.
- 1881 *Whitehead, WALTER*, F.R.S. Ed., Senior Surgeon to the Manchester Royal Infirmary, Manchester and Salford Lock Hospital, and Manchester and Salford Skin Hospital ; Professor of Clinical Surgery, Owens College, Victoria University ; 499, Oxford road, Manchester. *Trans.* 1.

Elected

- 1885 *Whitla, WILLIAM, M.D.*, Professor of Materia Medica and Therapeutics, Queen's College, Belfast; Physician to, and Lecturer in Medicine at, the Belfast Royal Hospital; Consulting Physician to the Ulster Hospital for Women and Children; 8, College square north, Belfast.
- 1852 *WIBLIN, JOHN*, The Hermitage, Clewer, Windsor.
Trans. 1.
- 1870 *Wilkin, JOHN F.*, M.D., M.C., Rose Ash House, South Molton.
- 1883 *Willans, WILLIAM BLUNDELL*, Much Hadham, Herts.
- 1896 *WILLIAMS, ALFRED HENRY, M.D.*, Rotorua, Harrow.
- 1859 *Williams, CHARLES*, Senior Surgeon to the Norfolk and Norwich Hospital; 48, Prince of Wales road, Norwich.
- 1887 *WILSON, ARTHUR HERVEY, M.D.*, 504, Broadway, Boston, U.S.A.
- 1889 *Wise, A. TUCKER, M.D.*, Davos Platz, Switzerland.
- 1850 *Wise, ROBERT STANTON, M.D.*, Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.
- 1879 *WOODWARD, G. P. M.*, M.D., Deputy Surgeon-General; 157, Liverpool street, Hyde Park, Sydney, New South Wales.
- 1892 *WRIGHT, ALMROTH EDWARD, M.D., Ch.B.*, Oakhurst, Netley, Hants.

ANNUAL MEETING.

March 1st, 1897, at 5 p.m.

WILLIAM HOWSHIP DICKINSON, M.D., President, in the Chair.

NORMAN MOORE, M.D.,
ROBERT WILLIAM PARKER, } Hon. Secs.

The minutes of the last Annual Meeting were read and confirmed.

The President nominated Dr. Rolleston and Mr. D'Arcy Power scrutineers of the ballot, and announced that it would remain open for an hour.

Mr. R. W. Parker (Hon. Secretary) read the Report of the Council as follows :

REPORT OF THE COUNCIL.

THE Council have to report that the year 1896-7 has been prosperous. Forty-eight Fellows have been elected, a larger number than in any previous year. Lord Kelvin has been elected an Honorary Fellow, and Professor Erb, Heidelberg, Professor Fournier, Paris, Professor Czerny, Heidelberg, Professor Koch, Berlin, Professor Laveran, Paris, Professor von Bergmann, Berlin, Professor Pierre Marie, Paris, Professor Kocher, Berne, Dr. Weir Mitchell, Philadelphia, Professor Gerhardt, Berlin, Foreign Honorary Fellows.

One Foreign Honorary Fellow, the well-known physio-

logist, Emile Henri Du Bois-Reymond, of Berlin, 11 Resident Fellows, and 14 Non-resident Fellows have died. The Society has lost 4 Resident Fellows by resignation.

An unusually large number of papers has been offered to the Society during the current Session. The number of papers received and not yet read is so great that it has not been found convenient thus far to set aside an evening for a special discussion. The attendance at the Meetings has been satisfactory.

The most important matter which occupied the attention of the Council during the past year was the replacing of the original 4 per cent. debenture loan by a new loan at 3 per cent. This was first brought under the notice of the House Committee by a letter from Mr. MacAlister, the Resident Librarian, dated March 23rd, 1896, and was followed by a letter from the Treasurers to the President, by whom it was brought before the Council on May 19th, 1896. A Committee was formed, consisting of the President, the senior Treasurer, and Mr. Holmes, the Chairman of the House Committee, by whom the necessary negotiations were carried out with the holders of the debentures, and with other Fellows willing to take up the new bonds. In these negotiations they were zealously and efficiently assisted by Mr. MacAlister, to whom the Council, on behalf of the Society, would now convey their best thanks.

The Honorary Treasurers report as follows :

“The present financial condition of the Society is satisfactory.

“The Council of the Society having had the whole of the premises and property belonging to the Society valued, the Honorary Treasurers are able, for, we believe, the first time in the history of the Society, to present a balance-sheet showing the actual position of the Society as regards its property and finances. (*See pages xci—xciii.*)

“It can now be seen that the debenture-holders have ample security for their money, for the

Society's premises alone, *without their contents*, are valued at £51,150, which is about £15,000 more than its gross liabilities.

“The main feature of the past financial year has been the raising of a fresh debenture loan at 3 per cent. and the paying off of the original one, which bore interest at the rate of 4 per cent. per annum. £35,800 was required for this purpose, but owing to the large number of holders of the original bonds who were willing to accept the lower rate of interest, it was only necessary to raise £11,150 to pay off those who were unwilling to leave their money invested at the lower rate of 3 per cent. This sum was quickly obtained, and the Honorary Treasurers had the satisfaction of completing the conversion of the loan on November 30th.

“The raising of the new loan and the paying off of the old one have led to some complications in the statement of the moneys received and payments made during the past year, and may render it difficult for the Fellows of the Society to compare the accounts as there shown with those of former years.”

STATEMENT OF RECEIPTS AND PAYMENTS.

“The total receipts during the past year amounted to £4264 6s. 9d., the total payments to £4808 9s. 5d.; this excess of payments over receipts is due to seventeen months' interest on the old debenture debt being paid in the past year; if the £564 17s. 4d. interest paid on November 30th be deducted from the total expenditure, it will be seen that the payments were within the receipts.

“The large balances which formerly were carried

over from year to year were, to some extent, misleading, for a half-year's interest on the debenture debt had to be paid on the first of January in each year, and the large sum required to meet it appeared as a balance in hand on December 31st of the preceding year.

"The Honorary Treasurers have to regret the loss of Mr. Swinny's services to the Society. He was the first accountant appointed, and by the order and method he introduced into the accounts rendered most valuable and lasting aid to the Treasurers. His place has been taken by Mr. Beavers.

"In conclusion the Honorary Treasurers wish to bring to the notice of the Council the valuable assistance they received from Mr. MacAlister, the Resident Librarian, during the past year. By his exertions the requisite money for completing the New Debenture Loan was speedily obtained, and by his forethought, care, and knowledge of the steps taken in raising the original loan, the conversion was effected at a very trifling cost to the Society."

W. S. CHURCH,
J. WARRINGTON HAWARD, } *Treasurers.*

The Honorary Librarians report as follows :

"The most important event of the past year was a recall of all the books which Fellows had borrowed from the library. Several years had passed since the last general recall, and it seemed desirable that the record of books borrowed should have its accuracy tested in this way. Many books were returned that had not been upon the shelves for years, and a fresh record of books issued dating from last summer was begun.

“Upwards of three thousand books were borrowed during 1896, and this number is exclusive of the larger number of books issued for use in the library. During the same period one hundred and seventy volumes were obtained on hire from Mr. Lewis, and this department of the library work continues to give satisfaction. Four hundred and twenty-nine volumes were added to the library during the past year, in addition to the volumes of journals, transactions, and other serials, books in continuation, &c.

“The Society is indebted to many donors for valuable gifts during the year. The presentations include some sixty volumes from the library of the late Sir John Russell Reynolds, presented by Lady Reynolds; a large number of duplicate journals from Dr. J. E. Squire, and considerable parcels of books and pamphlets from Dr. Lionel Beale and Mr. D’Arcy Power.”

SAMUEL GEE,
RICKMAN J. GODLEE, } *Hon. Librarians.*

The House Committee reports as follows :

“The Committee has met six times during the twelve months.

“The British Gynæcological Society has availed itself of the power of terminating its lease at the end of the first period of seven years, but it retains a portion of its former holding. The room thus left vacant has been for the present let at a rent of £50 to Mr. MacAlister, so that though he has been authorised by the Council to sub-let the Resident Librarian’s rooms till October 8th, 1897, he continues to reside as required by the Bye-laws. The Westminster Debating Society has ceased to be a tenant.

“A satisfactory settlement has been made with the

Obstetrical Society as to the sum due for electric lighting. Under the new agreement that Society will have its own main, and deal directly with the Westminster Electric Corporation.

“Some minor lettings have been effected, and the casual lettings of the meeting rooms have produced the sum of £67 15s. 6d.

“Mr. Swinny, the Accountant, has left, and Mr. Beavers has been appointed in his place. The premises have been maintained in good condition without any extraordinary outlay, and the service of the Society has been kept up on the former economical scale.”

T. HOLMES, *Chairman.*

The Committee on Climatology and Balneology has been engaged in the preparation of Reports on those parts of England and Wales which are not dealt with in the volume already published, and with Reports on the Climates and Mineral Waters of Ireland. Some changes have taken place in the constitution of the Committee during the past year. The President of the Society and Dr. Cheadle have resigned their seats, much to the regret of the Committee, and Dr. Bowles, Dr. Leech, Dr. J. W. Moore, Dr. Tooth, and Dr. Horton-Smith have been appointed Members. Dr. Horton-Smith has undertaken to share the duties of the Honorary Secretaryship with Dr. A. E. Garrod.

Mr. T. Pickering Pick, the Hon. Secretary of the Committee on Suspended Animation in the Drowned, reports that the work of investigation is proceeding but slowly owing to difficulty in obtaining material suitable for the investigation, *i. e.* young adult subjects free from lung disease, and in securing the attendance of the Committee on the necessarily very brief notice upon which he is constrained to summon the Members.

The Council has under consideration the revision of the Bye-laws of the Society. Any changes suggested will be submitted to the Society in due course.

The Council being desirous to be represented at the funeral of the late M. Pasteur, an Honorary Fellow, requested Mr. Jonathan Hutchinson, who was then President, and Sir Joseph Lister to undertake this duty. Sir Joseph Lister again attended on behalf of the Society at the second ceremony, on December 28th, 1896, when the remains were consigned to their final resting-place in the Pasteur Institute.

The Senior Treasurer submitted and explained the audited accounts.

The PRESIDENT moved—

“That the Report of the Council and the Treasurer’s audited Statement of Accounts be adopted and printed in the next Volume of the *Transactions*.”

Carried unanimously.

The PRESIDENT moved—

“That the change made in the Bye-laws by the Council, namely :—In Chapter XIII, section 2, line 2, the words ‘at least once’ have been substituted for the words ‘on the second Tuesday ;’ and the words on lines 3 and 4, ‘and except the Tuesday in Easter week,’ have been omitted, be and is hereby confirmed.”

Carried unanimously.

The PRESIDENT then delivered his ANNUAL ADDRESS.

The PRESIDENT declared the ballot closed, and called for the Report of Scrutineers. The Scrutineers reported that the following gentlemen had been duly elected :

President.—William Howship Dickinson, M.D.

Vice-Presidents.—William Marcet, M.D., F.R.S.,
James Edward Pollock, M.D., Frederick James Gant,
Charles Sissmore Tomes, F.R.S.

Honorary Treasurers. — William Selby Church, M.D., J. Warrington Haward.

Honorary Secretaries. — Norman Moore, M.D., Robert William Parker.

Honorary Librarians. — Samuel Jones Gee, M.D., Rickman J. Godlee, M.S.

Members of Council. — John Abercrombie, M.D., Robert Leamon Bowles, M.D., John Mitchell Bruce, M.D., David Ferrier, M.D., F.R.S., Joseph Arderne Ormerod, M.D., William Watson Cheyne, F.R.S., Henry Hugh Clutton, M.B., Frederic S. Eve, William Appleton Meredith, C.M., Robert Alexander Gibbons, M.D.

The PRESIDENT moved—

“That a vote of thanks be accorded to the Scrutineers.”
Carried unanimously.

On the motion of Mr. TIMOTHY HOLMES, seconded by Dr. GEE, it was unanimously resolved—“That the best thanks of the Society be given to the President for the Address just delivered, and that it be printed in the next volume of the *Transactions*.”

On the motion of Dr. C. THEODORE WILLIAMS, seconded by Mr. D'ARCY POWER, it was unanimously resolved—“That the best thanks of the Society be given to the retiring Vice-Presidents (Dr. John Harley, Mr. John Langton, and Sir William Mac Cormac), and to the retiring members of Council (Dr. Ewart, Dr. Semon, Dr. Turner, Mr. Barker, Sir William Dalby, Mr. Morgan, and Mr. Owen) for their valuable services to the Society during their terms of office.”

BALANCE-SHEET

LIABILITIES.	£	s.	d.	ASSETS.	£	s.	d.
3 per Cent. First Mortgage Debentures . . .	35,800	0	0	Freehold and Leasehold Property (As per valuation of Messrs. Giddy and Giddy, 2nd June, 1896).	51,160	0	0
Overdraft at Bank	159	16	2	Fixtures, Fittings and Furniture (As per valuation of Messrs. Phillips, Son and Neale, August, 1896).	1412	7	0
Sundry Creditors	890	10	8	Engravings (As per valuation of Mr. F. B. Daniell, 19th August, 1896).	555	0	0
Coupon Outstanding	19	4		Medical and Surgical Works in the Library (As per valuation of Messrs. Henry Sotheman & Co., 31st July, 1896).	7693	0	0
Balance, being surplus of Assets over Liabilities	25,005	9	3	Investment (New South Wales 4 per Cent. Inscribed Stock).	326	7	3
				Sundry Debtors for Rents	209	8	9
				Cash at Bankers. Coupon ac- count (unclaimed)	19	4	
				Cash in hand	509	13	1
					<u>£61,856</u>	<u>15</u>	<u>5</u>

NOTE.—The Society is also possessed of £641 16s. 11d. Consols, but as the sum in question is held in trust for a specific purpose, viz. the Marshall Hall Memorial Prize Fund, the capital sum has not been included amongst the assets of the Society.

Audited and approved,

TOM MUNDY,
Chartered Accountant.

13th February, 1897.

				Receipts					
				£	s.	d.	£	s.	d.
Balance 1st January, 1896 :									£
Cash in hand				218	11	4
„ at Bankers				692	7	4
									910
<i>Subscriptions, Fees, &c.:</i>									
414 Annual Subscriptions at £3 3s.	..			1304	2	0			
34 Annual Subscriptions at £1 1s.	..			35	14	0			
Composition Fees (Life)	..			63	0	0			
Entrance Fees		226	16	0			
							1629	12	0
<i>Transactions and Proceedings:</i>									
Sold by Messrs. Longmans	..			61	0	8			
„ Mr. H. K. Lewis	..			1	16	9			
„ Resident Librarian	..			13	19	8			
							76	17	1
<i>Rents</i>				2528	19	2
<i>Value of some lost books</i>					2	18	0
<i>Interest:</i>									
On Permanent Endowment Fund	..						12	12	4
									4250

£5161 1

W. S. CHURCH, M.D., }
J. WARRINGTON HAWARD, } *Treasurers.*

13th February, 1897.

Payments

	£	s.	d.	£	s.	d.
<i>Rates, &c.</i>				235	13	3
<i>Cleaning, &c.</i>				315	18	11
<i>Repairs, Alterations, Furniture, &c.</i>	196	8	7			
<i>Less</i> received for Obstetrical Society's Electric main, &c. ..	15	10	2			
				180	18	5
<i>Printing Expenses</i>				33	17	7
<i>Printing, Stationery, &c.</i>	167	0	11			
<i>Stamps and Telegrams</i>	16	14	1			
				183	15	0
<i>Salaries and Wages</i>				815	10	11
<i>Library:—Books and Binding</i>				207	8	8
<i>Transactions' and 'Proceedings'</i>				550	14	2
<i>Debentures:</i>						
<i>Do Debentures, redeemed</i>				100	0	0
Interest at 4 per Cent. (17 months)	1955	1	10			
Interest at 3 per Cent. on Deposits .. £32 2 7						
<i>Less</i> „ received from Bank; Deposits 13 16 10						
				18	5	9
Stamps on new Issue				18	11	6
„ for Transfers in connection therewith ..	29	16	6			
				2021	15	7
<i>for Valuations of Society's Property—</i>						
Freehold (Messrs. Giddy & Giddy)	21	0	0			
Library (Messrs. Sotheran & Co.)	5	0	0			
Furniture (Messrs. Phillips, Son & Neale)	5	5	0			
				31	5	0
<i>Printers' Fee</i>				10	10	0
<i>Post Charges, &c.</i>				10	17	3
<i>Committee: Copies of 'Climates and Baths' to Contributors and Committee</i>				19	2	3
<i>Miscellaneous</i>				52	14	5
				4770	1	5
<i>Amount overpaid on Debenture Account</i>	50	0	0			
<i>Less</i> Cheque for Interest outstanding	8	1	1			
				41	18	11
				4812	0	4
<i>Cash in hand</i>	509	13	1			
<i>Less</i> Overdraft at Bank	159	16	2			
				349	16	11
				£5161	17	3

Audited and approved,
TOM MUNDY, Chartered Accountant.

LIST OF PAPERS.

N.B.—THE Council of the Royal Medical and Chirurgical Society deem it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its *Transactions*.

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ADDRESS
OF
WILLIAM HOWSHIP DICKINSON, M.D.,
PRESIDENT,
AT THE
ANNUAL MEETING, MARCH 1st, 1897.

GENTLEMEN,—Though the hand of death has been heavy upon us, we have nothing else to regret in the course of the last year. Our losses in some respects have been irreparable, but numerically they have been more than made up by fresh entries, which have been unexampled in number, as have been the deaths which we have to record.

The great financial event of the past twelve months has been the conversion of the debt, somewhat after the manner of Mr. Goschen, whereby the Society will profit to the amount of £358 a year. I trust that this saving may be wholly utilised in the reduction of our indebtedness. Once clear we shall command the influence of wealth as well as of knowledge; we shall be able to spend liberally within the limits assigned to us, and assume a position in all respects worthy of the greatest medical society in the Empire. The lowering of interest, right and necessary as it was, could not be accomplished

without concession on the part of those who retained their investment, and inconvenience to those who renounced it ; but the Fellows in general have contentedly deferred their private advantage to the public good, and acquiesced in an arrangement which in the state of the Society and the money market it was impossible to avoid.

Another event in the past year was the dinner at the Hotel Cecil. This was attended by 137 Fellows and guests. Many of the seniors of the Society were present, including two who have since been taken from us, and among the guests were many who honoured this Society as the representatives of others. Some shortcomings were to be regretted which another time can be guarded against, but on the whole the function seemed to give pleasure, and it is to be hoped that it tended to the consolidation of the Society, and the increase of its popularity. I trust that this social attempt may be repeated and improved upon, and if it should become annual I think it would be to the benefit of the Society. Even if such a friendly gathering does no good it can do no harm, for no one need take part in it who does not wish to do so, and the cost to the Society is nothing.

I now have to revert to a difficult, and in some respects a painful part of my task. The death-roll has been, as I have said, unprecedented, and not only in number but in the distinction of those who have been taken from us. It includes 1 Honorary and 23 Ordinary Fellows, some whose loss will be greatly felt within these walls. It includes three past-Presidents, seven Fellows of the Royal Society.

It will be my endeavour to present as truthful an account of each of the departed Fellows as my information allows. If I dwell more on professional achievements than personal characteristics, it will be because it is more easy to ascertain what a man did than what he was. *De mortuis nil nisi bonum* is a somewhat restrictive rule by which I shall not hold myself bound, but shall rather follow the example of the humble biographer of a great

man, "nothing extenuate nor ought set down in malice." Unmixed and indiscriminate eulogy is uninteresting because necessarily untruthful or incomplete. It will be my duty to refer to the lives of many who have enjoyed the esteem of a most critical profession, which is in itself a certificate of character, and a protection against the ill-nature of the most candid friend. But there is eternal truth in the saying of Johnson, "A fallible being will fail somewhere." Among those of whom I have to speak are five who attained to great distinction, and were the worthy recipients of honours conferred by the State. But though the same ends, professional eminence and royal favour, were reached by all, the roads were widely different, "as many arrows, loosed several ways, come to one mark."

I have prepared obituary notices, often necessarily brief, of all our Fellows who have died since the 1st of last March, and of four whose lives terminated before that date, but whose deaths were not made known to the Society in time to allow of previous mention. I have not thought it right wholly to omit any, holding as I do that every one who remained until death a Fellow of this Society is entitled to a permanent place in its records. Not counting the hitherto unmentioned losses which occurred before last March, we have before us an unprecedented, and even an appalling, tale of mortality. I will not tax the patience of the meeting by reading what I have written of all, but there are some whose position in the Society and in the profession was such as to demand even somewhat extended notice at this time and in this place.

The list of deaths since the last annual meeting is as follows:

Honorary Fellow.

Emil Du Bois Reymond . December 26th, 1896.

Ordinary Fellows

(Arranged in the order of their decease).

Dr. William Sharp . . .	April 10th, 1896.
Dr. Thomas Charles Steuart	
Corry	May 20th, 1896.
Sir John Russell Reynolds . .	May 29th, 1896.
Sir George Johnson	June 3rd, 1896.
Dr. Charles Henry Ralfe . . .	June 26th, 1896.
Thomas O'Connor	July 7th, 1896.
Dr. Alfred Thomas Brett . . .	July 11th, 1896.
Dr. Henry Moore Bowman . . .	July 17th, 1896.
Peter Yeames Gowlland . . .	August 11th, 1896.
Paul Jackson	September 4th, 1896.
John Jones Merriman	September 8th, 1896.
Sir John Eric Erichsen	September 23rd, 1896.
Sir George Murray Humphry . .	September 23rd, 1896.
Dr. John Langdon Haydon	
Langdon-Down	October 7th, 1896.
Dr. George Harley	October 27th, 1896.
William Edward Stewart . . .	November 12th, 1896.
Dr. George Augustus Frederick	
Wilks	December 22nd, 1896.
William F. Butt	January 15th, 1897.
Dr. Edward Ballard	January 19th, 1897.
Sir Thomas Spencer Wells . . .	January 31st, 1897.
Dr. James Ellison	January 31st, 1897.
George David Pollock	February 14th, 1897.
William Smythe Crawford . . .	February — 1897.

The Fellows whose deaths occurred before March 1st, 1896, but have not yet been mentioned, are the following :

Edmund Charles Johnson . . .	January 3rd, 1895.
Richard King Peirce	February, 1895.
Robert James Wilson	February 16th, 1896.
Michael Henry Feeney	February, 1896.

It is my first duty to bring before the Society the death of one of our Honorary Fellows, *Emil Du Bois*

Reymond, who died at Berlin on the 26th of last December, at the age of seventy-eight. His name is known to the whole world of biological science, and his fame is too securely established to need any corroboration from us. It is not necessary in this place to dwell upon researches which have become incorporated in the accepted total of knowledge ; it may be enough to say that his observations in connection with animal electricity marked an epoch in physiology, and were an important means of promoting the electrical branch of medicine.

Notwithstanding his French name, Du Bois Reymond was a German by birth, by education, and in sympathy. To speak of him worthily I may do so in the words of Professor Burdon Sanderson, "Du Bois Reymond probably never made an incorrect observation or performed a faulty experiment." In him we have lost one of the most famous of the eminent men who have adorned this Society by accepting honorary rank in it.

Before dealing with more recent losses, I may revert to that of *Mr. Edmund Charles Johnson*, who died in January, 1895, but whose death did not come to the knowledge of the Society in time to be taken notice of in the obituary of that or the succeeding year. It seemed to me that Mr. Johnson's benevolent and influential life should not be without notice in our annals.

He was the younger brother of the late Mr. Henry Charles Johnson, the well-known surgeon of St. George's. Mr. Edmund Johnson was born in 1821 and educated at King's College and St. George's Hospital. He became a Fellow of the College of Surgeons, and was one of the last recipients of the Lambeth M.D., which was conferred upon him by the then Archbishop of Canterbury in virtue of an ancient privilege which was abolished by the Medical Act of 1858. Mr. Johnson found his life's work when, at the age of twenty-two, he became the travelling companion of the late Viscount Cranbourne, the eldest brother of the present Marquis of Salisbury. Lord Cranbourne was blind, and his interests, in which Mr. Johnson par-

ticipated, were chiefly in the amelioration of the condition of his fellow sufferers. After visiting with Lord Cranbourne the principal blind schools in Europe, Mr. Johnson devoted himself to works of charity in this connection. He was the author of a report on the blind which was presented to the House of Commons in relation to the Paris Exhibition, and was offered, and refused, the decoration of the Legion of Honour. He became associated with many schools and institutions for the blind, and was a member of a Royal Commission on the Deaf, Dumb, and Blind, and in that capacity visited most of the institutions in England, Scotland, France, and Germany, whose purpose had reference to those classes. He was Vice-President of the Paris Congress on the blind, for which he was created an Officer of the Academy of France. He held many other honorary posts having relation to similar objects, and was the author of many works bearing upon them. Outside what may be called his speciality, he performed many honourable functions ; among others he was Deputy Lieutenant for the Tower Hamlets, Chairman of Magistrates for Middlesex and Westminster, and Chairman of the Hanover Square Division of the County of London.

Spared by circumstances from the necessity of medical practice, his life was devoted to charitable purposes and public usefulness. For many years I knew him as a respected and public-spirited Governor of St. George's Hospital, and could relate instances in which he was of especial service to the hospital and to the staff, with which he was always in sympathy. His life was honourable in a double sense, not only in conduct but in position. He died suddenly of cardiac failure in his seventy-fourth year.

He will long be missed by a wide circle of friends, and at the benevolent institutions upon which his time and thought were so largely bestowed.

Richard King Peirce, who had retired long before his death and was latterly little known in this Society, was

born at Canterbury and received his medical education at St. Bartholomew's Hospital. After passing the College and Hall he went to India in charge of troops, but met with a severe accident on his way out, which necessitated his return. He presently set up at Madeley in Shropshire, and afterwards at Notting Hill, where he met with much success. He was at one time Surgeon for Women and Children at the Blenheim Street Dispensary. He gave up practice in 1882, and afterwards lived in the neighbourhood of Windsor Forest and at Maidenhead, and was well known as a follower of Her Majesty's Buck Hounds. In 1894 he went to the south of France in consequence of failing health, and died at Mentone in February, 1895, of pneumonia consequent on influenza, having just completed his seventy-second year.

Robert James Wilson was held in great esteem in the towns of Hastings and St. Leonards, where he practised, at first in partnership, latterly alone, for forty-two years.

He studied medicine at the Westminster Hospital. After obtaining the Membership of the College of Surgeons he became a Member, and afterwards a Fellow, of the College of Physicians of Edinburgh, at a time when these qualifications were held to entitle the possessor to the appellation of Doctor.

Mr., or Dr. Wilson as he was called, seems to have been a man of real excellence of character and great kindliness of disposition, and one who fully merited the confidence which was reposed in him not only by the residents of Hastings and St. Leonards, but by the strangers within their gates. He had a large practice, which included at times members of the Royal Family and other personages of distinction. He was widely benevolent, and was connected with several charitable institutions. He was of a retiring habit and never sought local offices; he accepted, however, that of Justice of the Peace for the Borough of Hastings. He was respected by the profession as well as by the public, and it may be said truly that his unobtrusive and unostentatious life was of more service

in his day and generation than that of many whom circumstances have placed in more prominent positions.

He died of pneumonia, which supervened upon heart disease, on the 16th of February, 1896, at the age of sixty-eight.

Michael Henry Feeny was born at Castlebar, Co. Mayo, and received his medical education partly in Dublin and partly at St. Bartholomew's Hospital. He belonged to an ancient Irish family who were dispossessed of their lands in the comparatively modern time of Queen Elizabeth, and never recovered their former position. Mr. Feeny had two brothers in the medical profession, both of whom, like himself, died early.

Mr. Feeny was for some time the resident medical attendant of the late Lord Decies, who had a great regard for him. After Lord Decies' death Mr. Feeny practised first at Les Avants, in Switzerland, then in Lancashire. He never had good health. His final illness was connected with an abscess of the brain, for which he underwent an operation in the Home of St. Thomas's Hospital. He died in February, 1896.

The life of *Dr. William Sharp* takes us back to the early days of the Society. He was born in 1805, and became a Fellow in 1840, when Sir Benjamin Brodie was President. He was still writing in 1892, so his spell of mental activity was long, and he left behind him some results which cannot but be permanent. He was born in the West Riding of Yorkshire, educated there and at Westminster School, and in the West Riding apprenticed. His medical education was continued at the United Borough Hospitals and in Paris. He set up at Bradford, became Surgeon to the Bradford Infirmary, and in that town acquired a large general practice. His bent was to Science, and he became known as a promoter of local museums, in connection with which work he was, in 1840, made a Fellow of the Royal Society. After lecturing on chemistry at Hull he removed to Rugby, and was there active in advocating the teaching of Science in the public

schools. In 1850 he gave up the office of Reader in Natural Philosophy which he had held in Rugby School, and devoted himself to medical inquiries. It is probable that unconnected as he was at this time with any hospital, his researches were little corrected by pathological observations. Among other systems he studied that of Hahnemann, and was eventually led to renounce the errors of legitimate medicine for those of homœopathy. Legitimate medicine forty years ago was not wholly a beneficent art, and he may have been wisely sceptical on the one hand, though perhaps he was too credulous on the other. In 1856 he received the degree of M.D. from the Archbishop of Canterbury, from which it may be inferred that that ecclesiastic was less solicitous for orthodoxy in medicine than probably he was in theology.

Dr. Sharp was a voluminous writer. Our 'Transactions' contain a paper by him on Necrosis of the Jaw, which was published in the year 1844, before he changed his creed. His later productions were chiefly in the advocacy of homœopathy. In a cursory examination of them I have been struck with the apparent lack of pathological knowledge and consequent superficial views of disease; and with the general use of the *post hoc propter hoc* argument—the patient took the medicine and got well, therefore he got well because of the medicine. This argument is not confined to any system of medicine, nor is it always fallacious.

It is impossible to doubt that Dr. Sharp was fully convinced of the truth of what he promulgated. He was an "earnest inquirer," to borrow a phrase which is sometimes applied to other matters, but earnest and honest inquiry does not lead all minds to the same conclusions, even though it starts from the same premises. His claim to be remembered rests on his early efforts in the dissemination of scientific teaching. He died last April at the age of ninety-one.

Of Dr. Thomas Charles Steuart Corry, of Belfast, a brief notice must suffice. He was the eldest son of the late

T. C. S. Corry, M.P., of Rockcorry Castle. Dr. Corry was in general practice in Belfast, had been a dispensary officer in the time of the cholera epidemic about half a century ago, was long a guardian of the poor, and appears to have been constantly their friend. I learn that he was a man of wide charity, and was held in much esteem in his locality. He possessed some literary accomplishment, had brought together a considerable library, and was the author of 'A Guide to the Scenery, Music, and Antiquities of Ireland,' and of a volume of 'Irish Lyrics and Poems.'

Living as he did at Belfast he was known little in this Society, though he attained the rank of Vice-President at the Obstetrical.

Dr. Corry appears to have been one of those men who are happily not uncommon in the branch of the profession to which he belonged, whose best memorial is in the gratitude of the poor, whom he so actively and generously befriended. He died at Belfast, in May, 1896, in his seventy-first year.

Sir John Russell Reynolds was the son of a dissenting minister, and the grandson of a court physician. He was born in 1828. Having determined to follow the profession of his grandfather, he proceeded in due time to University College. Here his course as a student was one of much distinction, but his means were small and he did not at first think of settling in London; he had relatives at Leeds, and there commenced practice. Soon afterwards, however, he was recalled to town under circumstances which must receive mention. Dr. Reynolds, as he then was, had attended the demonstrations and acquired the friendship of Dr. Marshall Hall. This great physician being about to retire, or partially retire from practice, made over to Dr. Reynolds his house, furniture, and equipage. It could not be expected that these possessions should be transferred without payment, and Dr. Reynolds was as well entitled as another to become the purchaser. But Dr. Marshall Hall

issued a circular to his patients, commending Dr. Reynolds to them, and proposing to maintain with him a consultative relation. Dr. Hall was a Fellow of the College of Physicians, with which corporation Dr. Reynolds had, at this time, no connection whatever. Dr. Hall's part in this transaction was severely censured by the College. The censure was probably accentuated by the unbecoming attitude which Dr. Hall adopted towards the College, declining to appear before the Censors' Board, when summoned, and addressing it in a disrespectful and offensive style. This censure was administered in the year 1853. In the following year Dr. Reynolds became a Licentiate, and was made a Fellow in 1859, almost as soon as was possible. It may be inferred from his early selection for this honour that the College held him free from blame, whatever may have been the case with Dr. Marshall Hall. I have been told that Dr. Reynolds did not gain many patients directly, from this transaction, but it nevertheless placed him as the accredited successor of the physician who held the first place in regard to disorders of the nervous system, and contributed largely to his future fortunes.

Between the years 1855 and 1865 Dr. Reynolds became in due gradation, Assistant Physician to the Hospital for Sick Children, Assistant Physician to the Westminster Hospital, Assistant Physician and full Physician to University College Hospital, and finally, on the relinquishment of the Chair by Sir W. Jenner, Lecturer on Medicine. Dr. Reynolds' lectures are spoken of as exceptionally good; graceful and admirable in form and expression, they could not fail to be; but they are represented as not only instructive but complete in instruction, so that a student has been known to take a high place in Medicine at the University of London who used his notes of these lectures as his only text-book. In 1869 Dr. Reynolds was made a Fellow of the Royal Society at the instance of his teacher, Professor Sharpey. To bring to an end this enumeration of what may be termed his minor dis-

tinctions, distinctions which at least convey less prominence than the crowning honour which came towards the close of his life, he was chosen in 1878 as Physician to the Household.

Gifted with remarkable literary powers, Dr. Reynolds was a writer of rare grace and elegance. He has left behind him much that was useful and much that was attractive. It was not given to him, as to few, to write for all time. He left no permanent stepping-stones in the path of science. There have been greater clinical observers, more profound physicians, and more original thinkers, but perhaps few who could clothe the knowledge of the time in more appropriate and attractive language. His genius was of the second order—expository, not creative. Most of his writings bore upon disorders of the nervous system, and tended to enhance his practice in this speciality. The most noteworthy was his book on 'Epilepsy' published in the year 1861. This was the result of much work. I am told that he repeated many of the experiments there referred to. When this was written I suppose it was the best treatise in English on the subject. I need not specify many other briefer writings, chiefly in the form of papers and addresses. In the latter he often displayed a rare felicity, elevating the subjects on which he touched and often rising into poetry and eloquence. Among the papers is one which appears to me to be less judicious than others, since it commends the treatment of rheumatic fever by perchloride of iron, based on a doubtful analogy between rheumatism and such infective diseases as erysipelas and diphtheria. If in rheumatism acid abounds and elimination is to be desired, the introduction of a drug which is at once acid and astringent seems scarcely indicated. It is easy to pick out one paper among many with which every one is not in accord, but for most of the productions of his pen it is not possible to feel anything but admiration. His genius was more literary and philosophical than scientific, and the admiration due rather to the writer and thinker than to the physician or pathologist. His largest contri-

bution to medical literature was the 'System of Medicine,' in five volumes. Whether because the editor was indulgent or the contributors dilatory, thirteen years were allowed to elapse between the issue of the first volume and of the last. The work is unequal, but contains many papers of great merit, and was a valuable compendium of the knowledge of the time. Dr. Russell Reynolds took part with his brother in writing a novel entitled 'Yes or No.' I have not had an opportunity of seeing this, but it is described as semi-theological, and not unlike Kingsley's 'Two Years ago.' Not only was Russell Reynolds a polished writer, but he was, to use a fashionable term, a man of culture. He was widely read, his knowledge of the poets was continually in evidence, he was familiar with French and German, he was a musician, and something of an artist.

One who knew him intimately thus writes of Sir Russell Reynolds:—"He had many of the qualities of a fine physician. He was an excellent critic, and saw quite clearly the imperfections and half-truths of all systematic presentations of medicine. I think he always had before him that the patient was more than the sum of his diseases. He had a great deal of sympathy, perhaps too much. He was very jealous of overbearing the personality of his patient, and in this respect I think he was better than the great physician who preceded him in the Presidential chair. Although he had great critical faculty, and could show up inconsistencies and quackery, as he did in his address on specialism, he never, I am sure, said anything ungenerous of a fellow-worker, and I know that in some notable instances he was magnanimous. We always felt that he had it in him to do a great deal more than he did had he had the stimulus of necessity, but what he did was of very fine quality."

The crowning glory of his life was the Presidency of the College of Physicians, which fell to him by a majority of only two votes over the present holder of the office. For this post Dr. Reynolds was fitted by nature beyond most men. His never-failing courtesy, his considerate bearing

to one and all, his graceful language and refined utterance, combined to confer upon him, so long as his health lasted, rare qualifications for the Presidential chair. The Fellow of the College who beyond all others is qualified to speak of Sir Russell Reynolds in his Presidential capacity makes much of his native courtesy and kindness, gentleness and geniality, and speaks of his striking tranquillity and composure of demeanour even under very trying circumstances. He refers to his eloquence and grace of style, which were apparent in his extemporaneous utterances as well as in his more studied compositions. As President he was tried both by failing health and by the pressure of exceptional and extraneous duties ; but there were occasions, such as the delivery of the Annual Address, when his peculiar fitness for the office was conspicuous. His Presidency was marked by one important event, and the active part he took in regard to it. He strenuously and successfully opposed the admission of women to the diplomas of the College, and thus, says my informant (with whom personally I heartily agree), " he saved the College from a great disaster."

The accession of Dr. Russell Reynolds to the Chair at the College was followed by a baronetcy.

From what I have said it will readily be inferred that he acquired the attachment of all who were brought into contact with him, professionally or socially. This contributed, as it could not fail to do, to the success which was primarily due to his writings upon nerve disorders at a time when the modern lights were still below the horizon.

I now have to record the loss of a prominent Fellow, once President of this Society, and for many years a conspicuous figure in the medical life of the metropolis, *Sir George Johnson*. He was born at Goudhurst, in Kent, in which town he was educated, and in which county he was apprenticed. At the age of twenty-one he entered at King's College in the Medical Department, and became much distinguished as a student both there and at the University

of London. Not to mention the early steps of his professional career, Dr. Johnson was in 1847 appointed Assistant Physician to King's College Hospital, a promotion which he had fully earned. He remained an active member of the staff until 1886, when he became Consulting Physician. He had held many offices in the Medical School,—Resident Medical Tutor, Professor of *Materia Medica*, Professor of Medicine and afterwards of Clinical Medicine, which last post was exchanged on his retirement for that of Emeritus Professor of the same subject. At the College of Physicians Dr. Johnson held almost all the appointments possible to him excepting that of President; and even as to that, a considerable minority of votes were recorded in his favour on the occasion of Sir Andrew Clark's election, a minority which would have been larger but for Dr. Johnson's obviously failing health. Among his many distinctions, apart from his hospital and the Royal College, it is necessary to refer only to the most prominent, the Fellowship of the Royal Society, the position of Physician Extraordinary to the Queen, and knighthood. But Sir George Johnson's distinction did not rest on the honours he received, but on the work he performed. He early gave evidence of his ability in his book on kidney disease, which was published as long ago as 1852. This aimed to elucidate the pathology of the kidney by means of the microscope, and was a great advance on anything that had been previously accomplished. When it was written many ways of examining the organ which have since been employed were unknown, and his results were necessarily incomplete, as his methods did little more than reveal the state of the tubes without adequately displaying their interstices. It is characteristic that he adhered to the last to the partial view of renal disease thus indicated, and re-asserted it in a small volume which he issued in the year of his death. He thought that the fibroid increase was not real but only apparent, the appearance being due to the atrophy of the tubes, not to the hypertrophy of what was between them. Whether right or wrong in this matter—and let it be said

that there are still some who think as he thought,—he made at a later date an important observation, as to the broad truth of which no doubt can pertain. He demonstrated the general thickening of the systemic arteries with the chronic granular kidney. This, in my opinion, was the best thing he ever did, and one on which his fame will securely rest. What Johnson advanced was always fated to excite discussion, and this was no exception. He regarded the arterial thickening as purely muscular; Gull and Sutton maintained that it was purely fibroid. The controversy was waged with obstinacy and even with acrimony, but both parties may now sleep in peace,—Johnson in the assurance that his muscular hypertrophy is established beyond question, and the champions of fibrosis with the knowledge that this too is recognised as a truth. At a late period of Johnson's life he frankly admitted to me, in reference to something I had written, that he then recognised the fibroid hypertrophy as well as the muscular. The permanent addition to our knowledge was in the discovery of the arterial thickening; there was room for discussion as to its nature and mode of production.

Together with renal questions, Dr. Johnson was deeply involved in controversy relating to cholera. While Junior Assistant Physician, in 1854, he introduced his castor-oil treatment on eliminative principles, and afterwards super-added a theory of cholera collapse, which he held to be due not to dehydration, but to spasm of the pulmonary arterioles under toxic irritation. It might be urged that thus to add castor oil to the cholera poison was but to give its meed of more to that which had too much; while the hypothesis of pulmonary spasm was not sufficing, since it attributed no part of the result to the changes in the blood necessarily produced by the discharges. As to why the pulmonary vessels, rather than the systemic, should be affected by the spasm, if spasm there be, it would be possible to urge, were one holding a brief for the pulmonary vessels, that if the poison be absorbed from the intestine and conveyed by the portal circulation, the pulmonary

vessels must receive it before the systemic. But this is not the time to discuss, but only to record. These views excited much opposition, which Sir George Johnson neither forgot nor forgave. As in the renal question, so with regard to cholera, he published a final re-assertion of his views just before his death. With this he gave a history of the controversy, in which he spoke of his opponents of forty years before with as much feeling as if they were foes of yesterday. He had none of that philosophy which is content to await the operation of time either to confirm or to correct. He did not resemble another Johnson who used to leave his assailants unnoticed, with the saying, "Depend upon it no man was ever written down but by himself." Beside renal disease and cholera, Johnson became prominent in many other matters, all of which displayed his activity of mind and many his militant temper. He was an early proficient in the use of the laryngoscope. He edited the fifth edition of Watson's inimitable 'Lectures,' and it is not to his discredit that he failed to do what was impossible—maintain the style of the original. Tests for albumen in the urine, the presence of sugar in it in health, the antecedents of Harvey's discovery, all occupied his attention and that of the medical papers. Among other disputations, he became involved in one with Sir W. Gull, on a point of etiquette connected with the Bravo case, which came before the College of Physicians, and was decided in Johnson's favour.

Sir G. Johnson was President of this Society from 1884 to 1886, and was one of the most important of our contributors, not only in number but in interest. Most of his matter which was novel, or involved aught that man may question, was brought in the first instance before this Society. His papers were often productive of others, and of discussion within and without. His opinions, theoretical as they sometimes were, were the result of much labour and thought, and he was apt to regard them as final. Towards those who did not accept his pronouncements his attitude was that of the orthodox in regard to heretics

in the ages of Faith. He brought into science something akin to the *odium theologicum* : here be truths—to reject them is to sin against the light.

In his hospital work he was much esteemed. An eminent physician who was his colleague during the greater part of his career writes thus :—"He was excellent as a clinical teacher ; was fond of his work, most regular in his attendance in the wards, kind and attentive to the patients, cautious in diagnosis, thoughtful and patient, popular and highly respected by his pupils."

I have been favoured with a careful estimate of Sir G. Johnson's worth by one who was his colleague in his early days and his friend to the end, one who assisted in his early renal work and witnessed the inception and progress of the cholera controversy. This judicious and accomplished physician—for such he was and is—dwells on Johnson's sympathetic way with the students, his power of enlisting their interest, and of entering into their difficulties ; upon his industry, conscientiousness, scientific spirit, logical faculty and power of expression, together with the ingenuity and inventiveness necessary for original investigation. My informant proceeds to say, "Of his sincerity and the reality of his convictions, in every case, I have no doubt, and I hardly think that anyone who knew him well could entertain any ; he believed intensely in what he taught, and he had all the courage of his opinions. This was especially apparent in the cholera controversy. That he honestly believed in the success of his treatment, and in the theory of cholera collapse he adopted and so vigorously maintained against all comers, I cannot doubt, and I cannot but admire the courage with which he defended it, single-handed, when it brought upon him a storm of ridicule and censure, and when his most trusted friends were advising him that he had little to gain and much to lose by what he was doing.

"There is no doubt that he possessed very considerable dialectical skill, and made the most of it. The ingenuity with which he met, and even turned to his own account

his opponents' points, was remarkable. His tenacity of purpose, and readiness to renew the contest on every fresh occasion, were scarcely less so. A controversial atmosphere was certainly not uncongenial to him. It has been suggested that Johnson fought, as the saying is, more for victory than for truth. I do not think so, though I can well understand that the intellectual combativeness to which I have referred may have gone far to produce that impression. It always appeared to me that when he had, often after prolonged investigation, convinced himself of the accuracy of his observations, and had with much thought and ingenuity framed an apparently logical and consistent theory or hypothesis to account for them, this last took so firm a hold on his mind (and the firmer, the more he had to defend it), that he became hardly in a position to estimate at their proper value any presentations of facts or views which might tell against or be irreconcilable with his own. So clear and consequential it all seemed to him, that he could hardly conceive any trained and intelligent person not seeing as he did, and had small patience, therefore, with some who thought and saw differently."

Sir George Johnson gave almost his whole mind to his profession. He had few interests outside it, though it may be mentioned that he was a keen sportsman, especially in the way of deer-stalking, in which he was successful even in his later days, when he suffered from paralysis agitans, an affliction which might have been thought to interfere with his skill as a marksman.

Sir George Johnson's tall and dignified figure, his handsome face and courteous manners, will long be remembered in this Society, while his works will claim attention long after his personality has been forgotten. His earnest, strenuous, and honorable life must ever command admiration and respect. What he did, and what he tried to do, the impetus which he gave to research, and the discussions of which he was the centre, have permanently modified the knowledge of his time, and will be

ever remembered in the medical history of the latter half of the nineteenth century.

He died on the 3rd of last June of an apoplectic attack which had seized him on the 1st. He lived his life to the last, and concluded it without suffering. With failing health and trembling fingers he stood to his guns to the last. On the morning of his fatal attack he had been characteristically, and probably happily, employed in writing a controversial article on cholera.

Dr. Charles Henry Ralfe, the son of a naval officer, was born in 1842. He received his medical education at the Bath United Hospital and King's College, London. After having been House Surgeon at the Lock, he entered at Caius College, Cambridge, and graduated with honours in Natural Science. He first came before the public as a general practitioner at Doncaster, but in 1869 he established himself as a physician in London. He soon obtained the appointment of Registrar at Charing Cross, and availed himself of the opportunities there afforded to work at Physiological Chemistry. His labours bore fruit in 1873 in the shape of a small but useful handbook on that subject. Shortly after this he became attached to St. George's Hospital as Demonstrator of Physiological Chemistry, and to the Seamen's Hospital at Greenwich as Physician. He used his special knowledge and his clinical opportunities in the investigation of scurvy, a disease which cannot be said, as yet, to have given up its secret, but which Dr. Ralfe threw light upon in pointing out the deficiency in it, not only of potash, but of the alkaline phosphates. He left St. George's and Greenwich on becoming in 1880 Assistant Physician to the London Hospital, which he continued to be until within a few months of his death.

On his resignation in consequence of failing health he was created Consulting Physician, I believe an unprecedented honour for an assistant. At the London Hospital Dr. Ralfe had given voluntary lectures on Physiological Chemistry, and had also lectured on Public Health. He was

useful in the School, popular with the students, esteemed and trusted by his colleagues, as it was his happiness always to be wherever he found them. Among these I may count myself, for I was associated with him when he taught at St. George's, and, at that time, acquired a liking and respect for him which endured to the end of his life.

Besides the work I have referred to Dr. Ralfe was the author of several others on cognate subjects, one on Clinical Chemistry, one on Urinary Pathology, and a more comprehensive treatise on Diseases of the Kidney. He contributed nothing to our 'Transactions,' but held the offices of Councillor and Referee. At the Pathological he was more active than with us; he frequently took part in the meetings as exhibitor and otherwise, and was a member of the Committee on Morbid Growths, and also of that on Pyæmia.

Dr. Ralfe died of phthisis, sequent on diabetes, on the 26th of last June, at the age of fifty-four. He was a type of the best kind of physician. He used his opportunities for advancing knowledge with ability and success, and without the purpose of an advertiser. He was cultivated and well-read, upright and honorable, kindly and personally attractive. His loss will be regretted by all who knew him.

Thomas O'Connor, of March, Cambridgeshire, died on the 7th of last July, at the age of eighty-three. He had an extensive general practice about this place, where he had been for more than half a century. He was devoted to his professional work, and throughout the fen country, I am told, his name was a household word. He was a man of excellent ability; he was a good classical scholar, and amid the distractions of country practice fitted himself for the Fellowship of the College of Surgeons. He was the author of several papers which are published in the 'British Medical Journal,' notably one upon ergot. He was hospitable and genial, and his society was much appreciated by his medical neighbours.

Dr. Alfred Thomas Brett, of Watford, was more than a

local practitioner ; he was, latterly at least, something of a public character. He was well known in Watford and Hertfordshire, not only in medical practice, but in connection with many matters of general interest and utility outside the profession of medicine. He was a local leader in all that concerned education and public health. He was actively associated in Watford with the Public Library, the Natural History Society, and the Endowed Schools, and held many appointments in the town, medical and non-medical, which are too numerous to name. He was a member of the Hertford County Council. He was prominent in connection with the British Medical Association, and with the Association of the Medical Officers of Public Schools. Within the latter society I often met him. He presented himself to me as one of a sort of which there are not too many. He was a doctor, and more than a doctor—a man whose wide sympathies and numerous points of contact with his fellow-townsmen and fellow-workers could not fail to extend the respect which pertained to his calling. Our profession is an engrossing one, and often occupies our thoughts to the exclusion of social demands and public interests. Dr. Brett was full of what may be termed local patriotism, and did what he could—and that was much—to benefit with the widest scope the community among which his lot was cast.

He died of Bright's disease on July 11th, 1896, at the age of sixty-eight.

In the death of *Dr. Henry Moore Bowman* a life of great promise was extinguished. Dr. Bowman was born in Westmoreland, and educated partly by his father, a clergyman, and partly by the natural objects which formed his surroundings. His professional instruction was received at St. Bartholomew's Hospital, where he was distinguished as a student, as he also was at the University of London. He was the author of several papers, one of which on "Diseases of the Spinal Cord," published in 'Brain,' deserves especial mention. At the time of his death he held the offices of Assistant Demonstrator of

Physiology and Pharmacy at St. Bartholomew's, and of Assistant Physician to the Royal Hospital for Diseases of the Chest.

He was, as I learn, a man of much thoroughness and accuracy, popular and successful as a teacher, and one to whom those who came within his scope were much attached. Had he lived a great deal might have been expected of him.

His end was sudden and unexpected. Having retired to rest in his usual health, he was found dead in his bed on the morning of July 17th of last year. The heart was found to be dilated and degenerated. He died at the age of thirty-one.

Peter Yeames Gowlland, whose decease I have now to refer to, was best known as for many years Senior Surgeon to St. Mark's Hospital for Fistula.

He was born in Kent in the year 1825, of a naval family. His father was a captain in the Royal Navy, of fighting renown, and his mother was the sister of naval officers. Mr. Gowlland received his medical education at the London Hospital, where he held the offices of House Surgeon, Demonstrator of Anatomy, Assistant Surgeon, and Lecturer on Anatomy. He was very successful as Demonstrator, and was assisted in that capacity by very considerable artistic facility. He seems to have been very popular among his hospital patients. He found his life's work at St. Mark's Hospital, soon after his election to which he quitted the London, and gave himself up to the speciality with which he had become associated. In this he soon obtained a considerable practice. His surgical ability is highly spoken of by those able to judge of it, and he was regarded as conscientious, painstaking, kind, and unselfish. He was an honest man, and deservedly trusted by those who sought his skill. Though not known as a writer he had much literary taste. He was a sportsman in many departments, and, I believe, was eminent as a fisherman.

Five years before his death he left Finsbury Square, where he had practised for forty years, and afterwards lived in partial retirement. He died on August 11th,

1896, of uræmic coma. He leaves the reputation of an upright man and a judicious and skilful surgeon.

Of *Paul Jackson*, though a resident in London, it is strange to say that I can learn little excepting that he was a Fellow of this Society for fifty-six years. He was a student at the Westminster Hospital. He practised formerly in Thayer Street, Manchester Square, and latterly lived in the Wellington Road, where he died on the 4th of last September at the age of eighty-two. He had long retired from work. I am told that his patients placed great confidence in him, but beyond that I have received no information.

John Jones Merriman was a type of the highest class of general practitioner. He was greatly respected medically and socially, and did much to enhance the respect due to his calling. He was the third lineal representative of a medical firm which existed at Kensington for 110 years under the name of John Merriman. Like others of his family he was educated at St. George's Hospital, where his course as a student was not without distinction. He was, like his father, associated with the Kensington Dispensary, of which he was an active supporter.

In 1853 he was appointed surgeon to the household of the Duchess of Teck, and latterly became general medical attendant to the Duke and Duchess. He retired to Worthing in 1894, where he died on the 8th of September, 1896, in his seventieth year.

His life was one of unobtrusive usefulness and unblemished honour. He leaves behind him no more worthy member of the branch of the profession to which he belonged.

Sir John Eric Erichsen was of mixed race,—his father a Dane, his mother English. He was born at Copenhagen in the year 1818, and was educated in England, mainly at University College. As a pupil of Sir Robert Carswell he was early indoctrinated with pathology. Soon after the completion of his studentship, which comprised a course of study in Paris, he was appointed Lecturer on

Physiology at the Westminster Hospital. His attainments in this science led to his appointment as Secretary to the Physiological Section of the British Association in the year 1844, and his selection, in conjunction with Professor Sharpey, to inquire experimentally into the process of asphyxia. His researches in this matter were rewarded by the Fothergillian Gold Medal of the Royal Humane Society. In the year 1848 he sought and obtained the post of Assistant Surgeon at University College, and two years later he found himself full Surgeon and Professor of Surgery. His rapid rise on the staff was due to causes with which he had nothing to do—the various quarrels and resignations which followed the death of Liston. His promotion to the Chair of Surgery may be taken as a testimony to the reputation he had acquired at the age of thirty-two. It has been said that in revolutions men live fast; and Mr. Erichsen's professional course was accelerated by the dissensions among which his lot was cast. In the year 1853 he published his great work on 'The Science and Art of Surgery,' which went through many editions, has been translated into many languages, has been re-edited by younger men, and still holds its place as perhaps the best text-book on surgery, certainly the most popular. The estimation in which it was held at the time of the Civil War in America was shown by the fact that the Federal Government had it reprinted and distributed to every surgeon in its service. However the Northern States may have profited from this dissemination of useful knowledge, it is said that neither the author nor the publisher derived any advantage from it. This work did much to establish the position and secure the practice of the writer in general surgery. He was the author of others which drew to him business of a special, and perhaps not of the most desirable kind. These were entitled 'Railway Injuries of the Nervous System' and 'Concussion of the Spine.' The term "railway spine" was of his invention. These publications placed him in the position of a recognised scientific

witness in railway cases. He was employed sometimes by those who sustained injuries, sometimes by those who inflicted them. I have no reason to suppose that he was necessarily biassed in favour of the side by which he was retained, but the situation is an unsatisfactory one, since it tends to make a man an advocate when he should be a judge. The scientific witness should be the servant of the court, not of the litigant; his object should be to promote justice, not to maintain a cause.

To trace his later course in detail would be little more than to register a series of honours. He filled most of the responsible offices at the College of Surgeons, culminating with that of President, which he attained in the year 1880. When placed upon the Council he was an ardent reformer, but, what is not uncommon, he became less liberal when in office. At the College he displayed, as I am assured by Mr. Trimmer, much business capacity. At the Parliamentary election of 1885 he became a candidate, in the Liberal interest, for the seat now so worthily occupied by Sir W. Priestley, that of the United Universities of Edinburgh and Aberdeen. Whether to his advantage or the contrary, he was unsuccessful. He served on the Royal Commission on Vaccination, was made Surgeon Extraordinary to the Queen, and was chosen as President of University College, an honour the greater because not necessarily conferred upon a member of the medical profession. His immediate predecessor was the Earl of Kimberley. He was created a baronet early in 1895, and died in September, 1896, at the age of seventy-eight, full of years and honours, and regretted by a wide circle of friends. His death occurred at Folkestone, after a paralytic seizure which supervened upon symptoms of angina.

Mr. Erichsen, as he then was, was President of this Society in the years 1879-80, and filled the office with the efficiency and dignity which were characteristic of him; but his contributions to the 'Transactions' were but two in number, and those of no great value.

Erichsen must be regarded as an eminently successful

man, and success is the stamp of the world's approval. It is true he was fortunate, circumstances worked for him ; but at least he was able to avail himself of his opportunities, and to fill with acceptance the high place to which he succeeded. He owed his success to a happy combination of good qualities rather than to pre-eminence in one. I cannot learn that he ever did anything to advance the science of surgery. If he was more skilful than others in the practice of it, it was not in the manipulative parts. He was, as I learn, good in diagnosis and judicious in advice ; not so good as an operator by reason of his defective sight. One of his great merits, as I am assured by an eminent member of his own school, was his continual readiness to accept and embody the surgical advances of younger men. He was, says one who knew him well, "a man of good abilities, a good example of the average London hospital surgeon." This verdict applies only to the practical surgeon ; he must have had much more in him than practical surgery to have been supremely successful as a writer, and to have become at last President of University College. His clinical lectures, I am told, were more remarkable for elegance of language than profundity of thought. A great writer on surgery, not unknown within these walls, thus speaks of Erichsen.

After referring to his book as the chief surgical textbook in the English language, and one which has been translated into most of the languages of the civilised world, he adds, "This was in itself a great achievement, and will long secure for him a high place in surgical literature ; but Sir John Erichsen was more than an author. He was a distinguished teacher in a school where many great surgeons had preceded him, and he showed himself capable of carrying on their traditions and filling the chair once occupied by Liston and Syme, and he held down to nearly the time of his death a leading position in London. He was not, it is true, one of those who mark out the path for themselves, and who lead the way to fresh conquests in the domain of surgery. But he possessed a

judgment which, in clinical questions at least, was sound and enlightened by long experience, a great talent for administration, wise and weighty eloquence, dignity of presence, and elevation of view. Hence he was well fitted for the leading position in a great school of surgery. Outside clinical surgery his judgment was not so trustworthy." My correspondent instances, as showing want of judgment, Erichsen's tract on 'Hospitalism,' in which he advocated the destruction of existing hospitals as "pyæmia-stricken," and the substitution of temporary constructions. But the adoption of Lister's methods has done away with this necessity, if it ever existed, and we may exult in the accomplishments of the present while we sympathise with the endeavours of the past. Nor does my correspondent consider that Sir John Erichsen's reputation derived much benefit from his writings on the subject of Railway Injuries, or from the acrimonious style of controversy which he adopted when his doctrines on this subject were challenged.

Sir John Erichsen had qualities which would have served him well in any way of life. Among other useful gifts he was a ready and fluent speaker, and could effectively support the opinions he held. His genial and kindly nature secured to him the attachment of all with whom he was brought into contact, and made him widely popular. He took great interest in those who worked under him, and was always ready to lend a helping hand to those who needed it. As long as his personality is remembered, it will be with feelings of affection and respect.

The life of *Sir George Murray Humphry* was a remarkable one. Beginning as a general practitioner without a practice, imperfectly educated, poor and unfriended, he became the most influential man in the University of Cambridge, converted an insignificant Medical School into one of the greatest in the world, and left behind him a transformation which promises to endure as long as any part of our present University system. He found a school

rather select than numerous ; he left one rather numerous than select. Where half a dozen men at their fullest muster walked the hospital with the Professor of Physic ; where physiology was untaught, and of the other sciences on which medicine is founded, the only one in which the University provided adequate instruction was botany, there is now, owing to the efforts of Sir George Humphry and Sir George Paget, a school of medicine which numbers about 300 entries a year, and which in the teaching of the fundamental sciences has no equal in England and no superior anywhere.

Mr. Humphry, not to anticipate his later designations, was born in 1820 at Sudbury, in Suffolk, and after a local education was, at the age of sixteen, apprenticed to Crosse of Norwich, and no doubt employed in the subordinate parts of surgery and general practice. In 1839 he proceeded to St. Bartholomew's Hospital, and after having gained a gold medal in Anatomy at the University of London, passed the College and Hall, and became legally qualified to practise at the age of twenty-two. In the same year he was, through the influence of Mr. Paget, then Curator of the Museum, elected surgeon to Addenbrooke's Hospital, where Dr. Paget, the brother of the surgeon, was on the staff. Mr. Humphry had never been House Surgeon or Demonstrator, and had but recently completed his third year at St. Bartholomew's when he thus found himself a hospital surgeon with the responsibility of capital operations. When he reached Cambridge he must have had much to learn, and it may be added that he had everything to earn. I am told that he had to borrow a small sum wherewith to purchase a horse which was to carry him on his daily rounds. He soon acquired a considerable general practice in and about Cambridge, and attached to his horse a dog-cart, on the back seat of which he used to crouch, protected, as well as might be, from the wind and rain. On his appointment to the hospital he and Dr. Paget obtained the permission of the Governors to give Clinical Lectures, which hitherto had

not been done, and Humphry delivered in addition systematic lectures on Surgery, a course of which was published in the 'Provincial Medical Journal.' In the year 1847 Dr. Clark, Professor of Human and Comparative Anatomy, made over to Humphry, as his assistant, the portion relating to the human subject. Accepted as a University lecturer, Humphry now entered at Downing, and became a University student. Some years afterwards I had the privilege of attending his lectures on the bones. These were the finest lectures on human anatomy I ever heard. The only discourses which could compare with them were those by Owen, at the College of Surgeons, on Comparative Anatomy. Humphry brought to bear upon his limited subject a wealth of illustration drawn from comparative anatomy and physiology, and a breadth of philosophic thought which made those dry bones live as if they had been revived by some miraculous touch.

While thus lecturing for Professor Clark, Humphry brought out his great book on the Skeleton, which was one of much labour and originality, and at once procured for him the Fellowship of the Royal Society. Between his arrival at Cambridge in 1842 and the appearance of his book in 1858, he must have done an amount of work of which few would have been capable. In the first place he had to maintain himself by a laborious and ill-paid general practice. During part of this time he had to prepare for University examinations in Arts and Medicine. Not only did he perform the ordinary duties of a hospital surgeon with more than ordinary energy, but he gave two courses of lectures annually, at first clinical and surgical, latterly clinical and anatomical. In addition he found time to produce the treatise to which I have referred, and make the numerous observations and dissections upon which it is based. All this he did under the frequent embarrassment of ill-health, for he was never physically strong. On the resignation of Dr. Clark in 1866, human anatomy was separated from comparative, and the Professorship of the former assigned to Humphry.

This he retained until 1883, when he resigned it, with its emoluments, in order to become Professor of Surgery without stipend, a generous act which helped to further the great object of his life, and bring nearer to completeness the Medical School of Cambridge.

He had long been gradually emerging from miscellaneous practice, and now held the position of the chief consulting and operating surgeon in Cambridge and its neighbourhood. His lectures as deputy had done much to popularise the study of medicine in the University, and as Professor he bent all his energies towards what was virtually the great work of his life, the development of the Medical School. It was owing to his influence that the Cambridge School was completely recognised by the College of Surgeons. In 1859 the Cambridge Anatomy was partially recognised, Medicine and Surgery not at all. When he became Lecturer on Surgery, the Cambridge teaching on Anatomy, Medicine, and Surgery obtained complete recognition, and the University was placed on a level as regards the College of Surgeons with the other great Medical Schools. He was active in getting the Colleges to admit the claims of Natural Science, and in demonstrating to the profession the comparative inexpensiveness of a University medical education. He took a leading part in the construction of the new school buildings, and in the establishment of the Museum, and was helpful in the foundation of the Professorships of Physiology and Pathology. While giving his due to Humphry the name of Sir George Paget must not be passed without grateful recognition, for what he did towards effecting the great transformation. As to the Museum, this was Humphry's hobby. He spent much time in it and much money upon it, and sought material far and wide. If he ever were unscrupulous it was in the acquisition of pathological specimens,—a form of immorality by which other collections beside that at Cambridge have been enriched.

It is unnecessary to follow his later career in detail.

He acquired, or had thrust upon him, almost all the honours possible in his position. He became a member of Council and examiner at the College of Surgeons, and would have been made Vice-President and President had he not thought these offices incompatible with his Cambridge work. He for a time represented the University on the Medical Council. He was President of the British Medical Association in 1881, President of the Pathological Society of London in 1891. He was made an Honorary Fellow of Downing, and a Professorial Fellow of King's. He received titles of honour from many universities, and gave many lectures and addresses at the instance of various learned bodies. In 1891 he received the honour of knighthood, but of all the designations to which he was entitled the one in which he took most pleasure was, as he once told me, that of Professor.

As an author Humphry was remarkable both for quality and quantity. All he wrote was good, and there was much of it. Beside his book on 'The Human Skeleton,' he wrote one on 'The Limbs of Vertebrate Animals,' and another on 'The Human Hand and Foot.' He was the author of a small treatise on the coagulation of blood in the venous system, a subject in which he had had painful experience, and also of a small volume on 'Old Age.' To our own 'Transactions' he gave nine papers, and therefore was among our most prolific contributors. I need make no mention of his minor publications, which were too numerous to recapitulate, but I must not omit to mention that he was editor of the 'Journal of Anatomy.'

Of Humphry as a surgeon it scarcely becomes me to speak. When I attended the Addenbrooke he was very busy excising knees, in which he took particular delight. We students, as youthful and confident critics, used to think that he sometimes performed this operation when it might have been avoided; and patients on admission were known to say, "Now, Dr. Humphry, I am not going to have my knee took out." But I will quit my

personal recollections, which are worth little, for the more valuable testimony of others.

A great surgical authority thus writes of Humphry :—
“ He was a good and successful surgeon. Part of his success was no doubt due, as he himself with becoming modesty pointed out, to the fact that his hospital practice lay mainly among agriculturists, with constitutions untainted by the debauchery and excitements of town life ; but those who followed his practice were best able to judge how large a part of it was due to his own care, ingenuity, and good judgment. How well planned his operations were, and how well his patients recovered, is shown by the fact that in those pre-antiseptic days many, if not most of the wounds were left simply exposed to the air, and healed kindly, without any dressing whatever. His worth as a surgeon can be well appreciated by a perusal of the nine contributions which he made to the ‘ Medico-Chirurgical Transactions.’ Humphry was one of the chief operators, after Fergusson, who advocated and extensively practised excision of the knee. His papers in our ‘ Transactions ’ (vols. xli and lii) had a powerful influence in recommending an operation which was at that time unduly decried by its opponents, as it was unduly exalted by its partisans—for Humphry writes not as a partisan (in fact, he was, he says, at first prejudiced against excision), and he makes no exaggerated claims for it ; but he shows by unanswerable results the good which it may do in appropriate cases. His practice was criticised at the time, and he was thought by some to have used the operation too indiscriminately ; but a careful perusal of these papers would, I think, modify this judgment.” My correspondent refers to a paper of Humphry’s in vol. lxii of our ‘ Transactions,’ as showing that he was the first English surgeon who successfully removed a tumour from the male bladder, and also that he then saw how much advantage might in many cases be derived from the supra-pubic method. My correspondent adduces Humphry’s last contributions to our

'Transactions' in 1890 and 1891 as marked by wide research, and as showing that age and success had not checked his ardour or diminished his interest in the pursuit of knowledge.

Humphry had a great contempt for what he called "messes"—various ointments and lotions which used to be applied to newly made wounds; his principle was to do what he had to do, and then leave the parts alone. He never took kindly to antiseptic surgery.

Humphry's personality will long be remembered. Who that knew him can forget his attenuated figure, his lean and starved look, his keen black eyes, and his hair as straight, and to the last as black, as the plumage of a raven? There was a fascination in his glance, so piercing and so inquiring; like Cassius, he seemed to look quite through the deeds of men. His consuming energy, his active mind, and his feeble frame irresistibly recall the description of the fiery soul which "o'er-informed the tenement of clay." He had no amusements, or rather his only amusement was travelling, and even with that his chief attraction was the hospitals and museums. He was penurious in all that concerned his own indulgence; but he was hospitable, and in large matters profusely generous. Having begun poor, he ended rich. All he became possessed of was the result of his own industry, and was made chiefly by the multiplication of small fees. Humphry was full of resource, and generally succeeded in getting his own way, whatever it was,—a measure of success which did not endear him to those who thought differently. But his aims were generally unselfish; they were seldom personal, but were directed to the development of the Medical School and the good of the profession.

He died at the age of seventy-six, of cancer of the bowel, having nearly to the last retained his customary interests and many of his customary occupations.

Dr. John Haydon Langdon Down was born in the year 1828, in the village of Antony St. Jacob, in Cornwall, where his father practised as apothecary. At the age of

eighteen he became a student at the Pharmaceutical Society in Bloomsbury Square, and there became proficient in botany and materia medica, and distinguished in chemistry. At the age of twenty-five he entered at the London Hospital, where, and at the University of London, his career was successful and even brilliant.

In 1858, having now passed the College and Hall, he was elected Medical Superintendent of the Earlswood Asylum, and there was introduced to what proved to be the work of his life. In 1859, now M.B. and a Member of the College of Physicians, he was appointed Assistant Physician to the London Hospital, the duties of which post were not held to be incompatible with residence at Earlswood. In the year 1868 he founded an Idiot Asylum of his own at Hampton Wick, under the name of Normansfield, which gradually assumed large dimensions, and where great numbers of the imbecile offspring of the upper classes were lodged and treated with profit, I dare say to themselves, certainly to the proprietor. With the responsibility of this great commercial undertaking, Dr. Langdon Down retained his position at the London Hospital, and was helpful in the School as Lecturer successively on Comparative Anatomy, Materia Medica, and Medicine. He duly rose on the staff until 1890, when he was, in fulness of time, eliminated as Consulting Physician. He thus lived a divided life, and "contrived a double debt to pay." The successful management of the Asylum was, I believe, largely due to Mrs. Langdon Down, and Dr. Langdon Down was enabled to perform his hospital duties without reproach, and even with credit. I learn from one of his colleagues that he was most punctual and conscientious in his hospital work, and much trusted and respected by those who worked with him.

In our Society he was Vice-President in 1890-91, and he gave two papers to our 'Transactions,' both bearing on "Congenital Deficiencies of the Brain." He was not a voluminous writer, but when he wrote it was generally with approval. He made nine contributions to the 'Patho-

logical Transactions.' He was the author of 'An Ethnological Classification of Idiots,' of 'Observations on the Mouth and Teeth in Idiocy,' of 'A Course of Lettsomian Lectures on the Mental Affections of Childhood and Youth,' and of other less considerable papers, mostly bearing on similar subjects. Dr. Langdon Down became old prematurely; in his latter years, though only sixty-eight when he died, his failure both of body and mind was conspicuous. His death, which was sudden, occurred at Earlsfield on October the 7th of last year.

Dr. Langdon Down was strikingly handsome. If, as has been said, "to be a well-favoured man is the gift of Fortune," this was the only respect in which Fortune favoured him. In early life he was poor, and owed everything he became possessed of to his own exertions.

Enough has been said to show that he was a man of high character, of much ability, industry, and power of organisation; one who might have done something considerable as a physician, had he not been encumbered with a pursuit in which medicine took only a subordinate part.

I approach the life of *Dr. George Harley* with a double duty; to do justice to his great gifts, and not to ignore his small failings. In him considerable talents and indomitable energy found expression in an enthusiastic assertiveness which did not always display them to advantage. His success was further hindered by disease, which cost him his hospital appointment, and in one shape or another accompanied him through the greater part of his life. The physical difficulties he had to contend against would have been insuperable to most men, but with him were to a great extent countervailed by his elastic and energetic temperament, and his determination not to be overcome. All who witnessed it must have admired the victorious struggle of mind over body, of mental force over physical failure.

Dr. Harley's life may be briefly related; his writings, adequately to consider them, would occupy a longer time than I can venture to devote to them. He was born at

Haddington, educated there and at the University of Edinburgh, and retained the characteristics of a Scotchman all his life. On leaving Edinburgh he went to Paris, and there worked for two years in the physiological and chemical laboratories of that city, and was there honoured with the Presidency of the Parisian Medical Society. While in Paris he discovered the presence of iron in the colouring matter of the urine, a discovery which was at first disputed, afterwards accepted. He spent the next two years at the German universities, under the direction, among others, of Liebig, Kölliker, and Virchow. He thus had a prolonged and complete scientific education, such as falls to the lot of but few. He devoted to physiology and chemistry four years, which most men with similar aims employ in gaining hospital experience in junior appointments. Almost immediately upon reaching London, on the conclusion of his foreign studies, he obtained the post of Curator at University College, then that of Lecturer on Practical Physiology and Histology, then that of Professor of Medical Jurisprudence, and finally that of Physician to the Hospital, which office was conferred upon him in 1861. In 1864 he presented to the Royal Society an elaborate research on the chemistry of respiration, which was rewarded with the Fellowship.

His career was interrupted by an accident which cost him two years of professional life and his hospital position. While working with the microscope as Demonstrator of Histology a vessel gave way in the left retina. This was followed by retinitis and glaucoma in the left eye, and sympathetic inflammation in the right. He was advised to submit to excision of the eye primarily affected, but reasoning as a physiologist rather than as a surgeon, he determined to have recourse to functional rest, to which end he shut himself up in a dark room for nine months, and came out with his eyes restored. This does not complete the tale of Dr. Harley's bodily impairments. He had been subject to gout from the age of sixteen; twenty-two years ago, after a tour through Lapland and

Russia, he was attacked with what was called rheumatic gout, which left him weak in the legs. Soon afterwards he fell down and broke the lower end of one fibula, which never properly united. But his power of walking remained more imperfect than this accident could explain, and it was thought by some that he had become the subject of locomotor ataxy. After death it was found that the paraplegic symptoms were due to pressure on the cord in connection with two carious lumbar vertebræ. How few men with such a record of ill-health would have done as much as Dr. Harley accomplished! Even within his dark chamber he dictated, through a partition, a book which was published and approved of.

Dr. Harley's bent was scientific rather than clinical, but he nevertheless was practically active, especially in regard to affections of the liver. But he by no means limited himself to this organ. Indeed, so numerous and so various were his topics that he was one of the most diligent inquirers, and one of the most productive writers of his time. Some of his best observations were upon the urine. So great was his range, including anthropology and spelling, that I cannot do more than bestow a passing touch on the more prominent of his performances. In the early sixties he was active at the Pathological Society, and was frequently selected as a referee when special skill in chemistry or microscopy was wanted. He strenuously controverted the views of Addison with regard to the supra-renal bodies, and brought forward observations and experiments to show that these organs might become diseased or be removed without any noticeable symptom. He justified his claim to speak with knowledge on this matter by obtaining in 1862 the Astley Cooper Prize, which for this year had reference to the structures in question. One of the most valuable of Harley's observations was, in my judgment, that upon Intermittent Hæmaturia, which was brought before this Society in 1865. Dr. Harley was the first to note the destruction of the blood-corpuscles in this condition, and the appearance

in the urine of the products of their disintegration, and thus to differentiate between this and other kinds of hæmaturia. He, however, attributed the disorder to hepatic disturbance, instead of, as was suggested at the time, and has since been generally accepted, to changes originating in the blood itself, or at least occurring there as the direct result of the malarial action. Dr. Harley's *magnum opus* in every sense was his work on 'Diseases of the Liver.' Of this it may be said that it would have been a greater book had it been a smaller one. Like everything he wrote, it is full of learning and research; but it is expanded with details, some of which might have been well omitted. Like his conversation, it is didactic and somewhat egotistical.

With regard to our Society, Dr. Harley, beside contributing the paper which has been referred to, served on three scientific committees, and held the office of Vice-President.

It may be said of him in final retrospect that few have done as much under so great disadvantages.

He died quite suddenly on the 27th of last October, in a manner which may be envied, from rupture of a coronary artery and hæmorrhage into the pericardium.

William Edward Stewart was probably better known to many of the Fellows of this Society than to me. He was born in 1821, and educated medically at University College Hospital. His working life was spent in general practice, first in Weymouth Street, latterly in Harley Street. He held several appointments in connection with benevolent institutions; he was Surgeon to the Marylebone Provident Dispensary, to St. Elizabeth's Home in Mortimer Street, and to the Trinity Home and Residence for Governesses; he was Medical Attendant to the Establishment for Gentlewomen during Illness, where he worked for some time with Miss Florence Nightingale.

In 1894 his failing health compelled him to leave London for Brighton, where his death occurred suddenly in November, 1896.

Dr. George Augustus Frederick Wilks was born in 1811. He was educated medically at Edinburgh, where he graduated. After studying in Paris he came to London and lectured on botany and materia medica, first at the Charlotte School of Medicine (about which school I have no information), and afterwards at St. Thomas's Hospital. He retired from practice in 1849, and after a few years went to Torquay, where he remained until his death. He was a prominent member of the Torquay Natural History Society, and was known as an author. He wrote a historical work on the Popes, and several essays on scientific subjects, mostly in opposition to the Darwinian theory.

He died on the 22nd of last December.

William F. Butt was well known, highly respected, and much employed as a general practitioner in Park Street, Grosvenor Square. Some years ago I used frequently to meet him socially, and, like most who knew him, held him in much esteem.

He was born at Gloucester in the year 1834. He received his medical education at the London Hospital. About five years ago his health broke down, in consequence, as was thought, of overwork, and he had to give up practice.

He died on the 15th of last January after a few days' illness.

Dr. Edward Ballard was one of a select class of men who render great service to the public, but are little recognised by them. The causes and prevention of disease occupied him rather than its cure.

He was born and bred at Islington, where a large part of his life's work was performed. University College was his *alma mater*, and the University of London the place of his graduation. At both he was distinguished. Early in life he became Physician to the St. Pancras Royal Dispensary, where he had as colleagues Dr. (now Sir Henry) Pitman, and the late Dr. A. P. Stewart. Later he became Lecturer on Medicine at the Grosvenor Place School, a private establishment belonging to the late Mr. Lane,

which was ultimately absorbed into the School of St. Mary's Hospital. The course of Dr. Ballard's life and his field of usefulness were determined in 1856, when he was elected Officer of Health for Islington on the creation of the office which he held. He retained this appointment together with private practice for sixteen years, at the end of which time he renounced both for the service of the Local Government Board, the Privy Council, and Sir John Simon. He received the Fellowship of the College of Physicians in 1872, and that of the Royal Society in 1889.

Before he became specialised he wrote a book on 'The Physical Diagnosis of Diseases of the Abdomen,' and another on 'Materia Medica and Therapeutics.' He was the author of many papers, five of which are in our own 'Transactions,' of a Prize Essay on Vaccination, and of many contributions to the science of public health. It was in connection with the latter that his most noteworthy work was done. He is believed to have been the first to trace the infection of typhoid to milk. He investigated an outbreak of diphtheria at Islington, the adulteration of butter with animal fats, trade effluvium nuisances, and the causation of summer diarrhoea. The last inquiry was his most extensive and laborious, and he was occupied upon it within a few days of his death.

He died on January 19th of this year, at the age of seventy-six, after a brief illness from bronchitis.

I cannot conclude this inadequate notice better than in the words of Sir John Simon: "My impression is that in times long after our own Dr. Ballard will be recorded as one of the chief confirmers and extenders of the sanitary science of his age."

The name of *Sir Thomas Spencer Wells* will ever be remembered in the history of the surgery of the nineteenth century. Though he was by no means a man of one idea, or a surgeon of one operation, yet, as is well known in this Society, it is upon one operation that his fame rests.

He was born at Hertford in 1818. He learned the rudiments of his profession from a general practitioner at

Barnsley, and afterwards became unqualified assistant to a parish doctor at Leeds. Here he was admitted to the lectures and the practice of the great school of surgery in this town, and in later life looked back with gratitude to the teaching of the second Hey and the elder Teale. From Leeds he proceeded to Trinity College, Dublin, and thence to St. Thomas's Hospital. On passing the College he entered the navy as assistant surgeon, and for nearly six years did duty in the Naval Hospital at Malta. He then left the navy, and after an interlude in Paris set up in London. In 1854 he became Surgeon to the Samaritan Free Hospital for Women and Children, then an institution of no great pretensions, in Seymour Street. Spencer Wells while in Paris had discussed the operation of ovariectomy with Dr. Waters, afterwards of Chester, but never witnessed its performance until, in the year of his becoming connected with the Samaritan Hospital, he saw it done by Mr. Baker Brown with, as was usual at that date, a fatal result. Not yet did Spencer Wells enter upon his destined path. In the same year, on the outbreak of the Crimean war, he temporarily resumed his position as a naval surgeon, and in that capacity proceeded to the East. In 1857, the year following his return, he was made Lecturer on Surgery at Lane's School, known as the School of Medicine, adjoining St. George's Hospital. At about the same time he became editor of the 'Medical Times and Gazette,' which post he retained for seven years. In the same year he made his first attempt in the speciality to which his future life was chiefly devoted. This was unsuccessful, but was followed in 1858 by a successful operation of the same kind. From this time for many years Spencer Wells was the accepted ovariectomist. He operated with a previously unknown average of success, though of late years, owing to the introduction of antiseptic methods, his average has been greatly improved upon. In our 'Transactions' for 1863 he published an account of his first 50 cases with, to apply justly a now discredited word, 33

cures. In a later volume, that for 1881, he gave a *résumé* of 1000 cases with 769 recoveries. Thus the earlier series gave 66 per cent. as the proportion of recovery, the entire series a proportion of 76 per cent. Spencer Wells had at first to encounter much opposition. The operation was passionately denounced by Dr. Robert Lee, and looking at its results before the time of Spencer Wells, the attitude of that honest and humane, if somewhat conservative physician was not unjustifiable. Dr. West, who had formerly been an opponent of the operation, became a supporter of it as performed by Spencer Wells.

The fame of Spencer Wells was enhanced by a book on 'Diseases of the Ovaries' which he published in 1865, which was modified and republished, and took its final shape in 1882 under the title of 'Ovarian and Uterine Tumours; their Diagnosis and Treatment.' This was translated into many languages, and acquired a more than European reputation. As a writer Sir Spencer Wells was clear and forcible. Though not a fluent or eloquent speaker, he could speak with effect when he had anything to say, which was as often as he rose to speak.

Honours and wealth accumulated. He was made Surgeon in Ordinary to the Household. He became President of the College of Surgeons in 1882, and a baronet in the succeeding year. He was made Foreign Associate of the Academy of Medicine of Paris, and received titles of honour from the Kings and Queens' College of Physicians of Ireland, and the Universities of Leyden, Bologna, and Charkof, and was made a Knight Commander of the Norwegian Order of St. Olaf. In our own Society he was Vice-President in 1881, and he contributed to our 'Transactions' as many as fourteen papers.

Sir Spencer Wells had an attack of influenza three years ago when travelling in India, after which paralytic symptoms slowly developed, and his broken health became painfully apparent. He was nevertheless able to attend the dinner of the Society on the 28th of last November,

and displayed gratification when some allusion was made to his ovarian exploits. Two months ago he went to the south of France. On the 31st of January, at Cap d'Antibes, he had an apoplectic seizure which proved fatal in twelve hours. He died three days before his seventy-ninth birthday.

An eminent surgeon, to whom I have more than once had to confess my obligations, thus writes of Sir Spencer Wells:—"As a surgeon Spencer Wells must in any ordinary circumstances have achieved distinction, for he had the love of his calling, the prudent boldness, and the capacity for careful attention to detail, which are the chief requisites for success. He had also seen much of practice under various conditions and in many countries. But it was the fortunate accident that directed his attention to ovariectomy which raised him to the highest rank in the profession, and enabled him to render services to humanity which no one in our day has surpassed with the single exception of Lord Lister. Now in speaking of Spencer Wells's career there is one error very commonly committed. Seeing the striking success of ovariectomy, and the immense saving of life it has effected, people often speak of him—the true founder of ovariectomy—as if his merit had been to combat, and by indomitable perseverance to uproot, an unfounded prejudice. We who are old enough to recollect the state of things at that time know well enough that it was no unfounded prejudice which great surgeons like Lawrence, and great obstetricians like Robert Lee, entertained against ovariectomy in these conditions; but a very real and well-founded objection, an objection founded on what was then the appalling mortality of the operation. And Spencer Wells's merit was not merely that by courage and perseverance he outlived a determined opposition, but that he so improved the details of the operation as to render it no longer murderous; and that by never operating in his hospital without professional spectators, and carefully publishing every case, he stopped the mouths of those who

believed that his apparent success was due to concealment of bad cases, till at length, when the cases counted by hundreds, and when his scholars began to attain the same success, it was no longer possible to deny to ovariectomy a place in ordinary surgery. This merit is far beyond any that could be attained by mere courage or perseverance, amply as he was endowed with those qualities. It was the reward of skill in diagnosis, and an operative dexterity only equalled by his boldness and his care. He was fortunate, indeed, in living long enough to enjoy his well-won honours, and to see ovariectomy introduced into every country in which scientific surgery is practised."

It may be thought that with the additional safeguards of recent times ovariectomy must have become common and successful, even though Spencer Wells had not shown the way. But he made it both, though he had not the advantages which modern science has provided. In him we have an illustration of the success which may be achieved by a man who does one thing and does it supremely well. We see the advantage of specialism. When a difficult and dangerous thing has to be done, it is better that it should be done by one who has had practice than by one who is seeking to acquire it. Spencer Wells was more fortunate with his later cases than with his earlier. The apprentice may be as confident as the master, but he will not be equally successful. In the surgical history of our time the name of Spencer Wells will ever retain a prominent and honorable place.

In *Dr. James Ellison*, of Windsor, the profession has lost a man who did much to increase the esteem with which it is regarded.

Dr. Ellison was born in India, and was educated in medicine at first by Dr. Thomas Walker of Peterborough, and afterwards at St. Bartholomew's Hospital and the University of Heidelberg. After having graduated at the University of London, Dr. Ellison began practice in Wimpole Street, but after six years he joined the late Mr. Henry Brown of Windsor, who was Surgeon-Apothecary

to the Royal Household, to which office Dr. Ellison ultimately succeeded. He also became Surgeon to the Windsor Royal Infirmary. Dr. Ellison's subsequent life was passed at Windsor. He died there of cancer of the œsophagus, on the 31st of last January, at the age of seventy-nine.

He was a many-sided and even a remarkable man. I learn that his professional accuracy and tact were such that he enjoyed in an unusual degree the confidence of his patients. He was honoured with the approval of the Queen, who, upon being informed of his death, commanded Sir James Reid to express to Dr. Ellison's family "Her Majesty's sincere regret at the loss of one who has served her so long and so faithfully, and for whom she entertained the greatest regard."

But not only in the profession was Dr. Ellison accomplished. He was widely read, a linguist, a musician, and an artist, and was familiar with the use, for scientific purposes, both of the microscope and the telescope. He was in the early part of his life fond of field sports, and was an active volunteer, first as a combatant, latterly as a medical officer.

In every way he maintained the honour of his calling, and by his cultivation and character acquired respect in more modes than often falls to the lot of one who belongs to a profession so exacting as that of medicine.

I now have to refer to the loss of one whose friendship I enjoyed for more than forty years, and whose recent death will be present in the minds of all who hear me speak.

George David Pollock was the second son of the great general who retrieved the disaster of the Khyber Pass. The general was one of four brothers, three of whom attained to great distinction, and rendered great services to the State. One became Chief Baron, another President of the High Court of India.

Mr. Pollock was born in India in the year 1817, and became a student at St. George's in 1837, with which

hospital he remained closely connected until his death. He was house surgeon under Sir Benjamin Brodie, and was subsequently sent by him to Canada to take medical charge of Lord Metcalfe, the Governor-General, who had become the subject of cancer of the face. Mr. Pollock was charged with the instructions of the great surgeon with regard to Lord Metcalfe's disease. Upon Mr. Pollock's return to England he became Demonstrator, and afterwards Lecturer on Anatomy. His first appointment as a hospital surgeon was to Great Ormond Street in 1852. In the following year he succeeded to the post of Assistant Surgeon to St. George's, and remained an active member of the staff of that hospital until his resignation in 1880. But his interest in the hospital and school did not terminate with his responsible appointment. He continued to take a prominent part as Governor, and as a member of some of the most important committees, in the management of the institution ; and as lately as October, 1895, he delivered, as he had once done before, the Introductory Address. To revert to matters outside the hospital, Mr. Pollock, upon the marriage of the Prince of Wales, received the appointment of Surgeon in Ordinary to the Prince. At the Pathological Society, of which he was one of the early supporters, he became Secretary in 1850 and President in 1875. He was chosen as President of our own Society in 1886. He long held the office of Examiner in Surgery for the Army and the East India Medical Service. No doubt the highest offices at the College of Surgeons would have been open to him had he not until the last year of his life refused to become a candidate for the Council. He thought it derogatory to solicit vouchers of his fitness as councillor. At the last election, hoping thereby to assist what is called the liberal party, he overcame his objection and complied with the necessary preliminaries ; but his advanced age, I presume, prevented his being successful. His attitude at the College always struck me as not what might have been expected from one of his natural bias. He was by nature conservative and conventional. He

stood by the old roads, and viewed any departure from them with distrust. But the old roads were never trodden with more dignity and propriety than by the high-minded and honorable gentleman whose loss we have now to regret. He even carried his conservatism into small particulars, and regarded with disapproval any innovations in dress or adornment which might take the undisciplined fancy of a generation younger than his own. Mr. Pollock's standard of professional conduct was high, even to fastidiousness. Anything approaching self-advertisement was abhorrent, and even impossible to him. Upright in conduct and punctilious in demeanour, he acquired the respect of all who value rectitude of purpose and the attitude of a gentleman. Mr. Pollock was born to social influence, and his kindly and sympathetic nature made him widely popular. I suppose few men have had more friends ; he took pleasure in extending a helping hand to those who wanted it, and the name of those whom he befriended was legion. Many must look back to him as having provided them with the first step in their success in life.

Much as I respected Mr. Pollock in every phase, I feel that my own knowledge is insufficient to do adequate justice to his accomplishments as a surgeon. I have therefore appealed to a surgical colleague, whom I will not indicate further than to say that I have already been indebted to him on this occasion. He writes, " Mr. Pollock occupied a very high place in the surgical profession. He was peculiarly well qualified both for hospital and private practice. He was a bold and skilful operator, a careful and sagacious consultant, and he was fond of teaching, so that he was equally acceptable to the students and his colleagues. He was endeared to his patients by a genuine kindness both of manner and of action, which especially fitted him for private practice, in which he early attained considerable success, and where he earned the gratitude and affection of a large circle of friends. Without any claim to originality, he worthily supported the reputation of the great surgical school at

which he was educated; and the hospital of Hunter, Brodie, Cæsar Hawkins, and Prescottt Hewitt counted him as one of its chief ornaments. To that hospital he was sincerely devoted, and its maintenance and improvement were the objects of his unceasing care and study. He had many other tastes apart from his profession, was fond of country pursuits, of farming, and of building, and found a refuge in his country seat near Ascot, which provided him with all the distraction he required, and enabled him to dispense with the holidays in which most London surgeons find it necessary to indulge. But, indeed, he had a genuine love of London, and of London practice, which mingled strangely with his enjoyment of long voyages and foreign travel on the rare occasions when he could persuade himself to take a long period of rest and change.

“Mr. Pollock was a vigorous supporter of our great medical societies. He served the Pathological Society zealously as Secretary, and presided over it with his usual ability. His services to our own Society in various minor offices, and as President, are too fresh in our memory to need further notice or praise from me. His contributions to our ‘Transactions’ were not numerous, but some of them at any rate are of high surgical interest. Mr. Pollock had given great attention to the cure of congenital fissure of the palate, and his paper in the thirty-ninth volume of the ‘Transactions’ marks the advance in that branch of surgery which his labours and those of Mr. Avery (to whom he does ample justice) had gained previous to the employment of chloroform in the operation and its consequent application to young children. The treatise also “On Dislocation of the Os Calcis and Scaphoid from the Astragalus,” in vol. liii, is a standard authority on that subject.

“I cannot close this brief reference to a long and honorable career without some expression of the deep regret which all St. George’s men must feel at the loss of

one so greatly esteemed as a teacher, a colleague, and a friend."

I need add but little in my own person to what has been so judiciously expressed. It always seemed to me that with Mr. Pollock's intellect and opportunities he might have done more than he did; that he might have left more footprints on the sands of time than he placed there. Perhaps he wanted energy. His brain was an excellent instrument, but the driving power did not seem commensurate. It may be that he was too successful to care to be more so; it may be that he was unduly sensitive to criticism, though on that score he need have had no apprehension.

Mr. Pollock died on the 14th of last month, in his eightieth year, after a few days' illness from pneumonia. He continued in practice until arrested by his fatal illness.

If honour, respect, and troops of friends are the proper accompaniments of old age, he had his due in these particulars.

William Smythe Crawford, of Liverpool, died in February of the present year, at the age of thirty-seven. He was educated at Cambridge, Liverpool, and Edinburgh, and became Assistant Surgeon to the Liverpool Cancer and Skin Hospital. He was the author of several papers dealing, among other subjects, with carcinoma, epithelioma, and sarcoma. He appears to have been much respected and regretted.

SPECIAL GENERAL MEETING

HELD IN

THE SOCIETY'S HOUSE, 20, HANOVER SQUARE, W.,

on Tuesday, June 8th, 1897, at 8.30 p.m.

W. HOWSHIP DICKINSON, M.D., President, in the Chair.

NORMAN MOORE, M.D.,
ROBERT WILLIAM PARKER, } Hon. Secs.

Present—30 Fellows.

The President on behalf of the Council proposed—

“That the Bye-laws as revised by the Council be adopted as the Bye-laws of the Society.”

Dr. Church explained that since the reprinting of the revised Bye-laws the Society's solicitors had suggested verbal amendments which, with the approval of his colleagues on the Bye-laws Revision Committee, he desired to formally move.

Dr. Church then proposed the following amendments in the Bye-laws now submitted for adoption :

Chapter II, sections 1 and 2 :

That for the words “Christian and surname” the words “full name” be substituted.

Chapter II, section 3 :

That the words “According to the form No. I in the Appendix” be omitted.

Chapter II, section 6, line 4 :

That the words “According to Form No. II of the Appendix” be omitted.

Chapter II, section 6, line 5 :

That the word “the” be substituted for “an,” and that the words “According to Form No. II of the Appendix” be omitted.

And that the Appendix be removed from the Bye-laws and incorporated with the Standing Orders.

These amendments were carried unanimously.

Dr. Abercrombie moved—

Chapter II, section 6, line 3 :

For the words " his " and " him " read " their " and " them."

This amendment was carried unanimously.

Mr. T. Holmes moved as an amendment that—

"Those portions of the revised Bye-laws which altered the radius of Resident Fellowship from fifteen miles to seven miles be not adopted."

The amendment was seconded by Mr. Haward, and was not adopted.

The President then proposed from the Chair the adoption of the revised Bye-laws as amended. A ballot was taken with the following result :

For	22
Against	2
					—
Majority	20

The President thereupon declared that the Bye-laws had been duly adopted in accordance with the provisions of the Charter and Bye-laws, and declared the Special General Meeting closed.



LOCOMOTOR ATAXY OCCURRING IN A YOUNG WOMAN

TABETIC ARTHROPATHY WITH DISLOCATION OF BOTH HIPS;
SIX PREGNANCIES OCCURRING IN THE COURSE OF
THE DISEASE

BY

THOMAS WILSON, M.D., B.S.LOND., F.R.C.S.ENG.

(COMMUNICATED BY DR. W. R. GOWERS)

Received April 11th—Read November 10th, 1896

LOCOMOTOR ATAXY accompanied by arthropathy of the hip-joints and occurring in a child-bearing woman is sufficiently rare to warrant the detailed record of a case. The following patient has been under observation for two years, in which time two pregnancies have been passed through. Previous to the time of coming under my notice the woman was under the care of Dr. Wade and of Mr. Barling at the Birmingham General Hospital. During the last two years the affection of the nervous system, although there have been vicissitudes, has remained nearly stationary, so far as can be determined clinically.

Mrs. A—, æt. 29, came to the General Hospital, Birmingham, on March 13th, 1894, complaining of pains about the lower abdomen, the sacral region, and across

the hips, and that her urine had been dribbling away since the beginning of January. The last menstrual period had ceased on January 2nd. On examination there was found remarkable deformity about the hips and loins, as detailed below. Abdominal examination showed the bladder to be distended, the organ reaching the umbilicus and forming an oval, moderately tense tumour, with long axis directed upwards and to the left. *Per vaginam* the cervix uteri was found low down and in the axis of the vagina; the body of the uterus, also in the axis of the vagina, was enlarged, rounded, and softened, and the long axis of the uterus ran to the right as well as backwards towards the hollow of the sacrum. The plane of the pelvic brim looked almost directly forwards. A little more than a pint of slightly turbid urine was withdrawn by catheter, and the uterus was then easily returned to its natural position. The diagonal conjugate diameter measured four and a quarter inches.

On March 16th the patient reported herself as feeling very much better. There had been no trouble with micturition since the previous visit. The uterus was now entirely above the pelvic brim, and extended as high as the umbilicus; it was as large as is usual at the third month of pregnancy, and was displaced bodily to the right, its long axis lying altogether on the right of the median line, and running somewhat obliquely from above downwards and towards the middle line. On vaginal examination the os uteri was found to be high up, and the cervix partly softened. There had been no vaginal discharge.

On this date, March 16th, a careful examination was made, and notes taken on which the following history is based. The patient had previously been in the General Hospital on two occasions,—in October, 1890, under Dr. Wade, and in July, 1891, under Mr. Barling; advantage was taken of the notes made on these occasions to control the woman's statements, and these were found to be remarkably accurate in almost every

detail. Until the age of sixteen, the patient was always very strong, and there appears to have been no infantile disease of importance. About sixteen there began to be shooting pains in various parts of the body, but especially in the lower limbs, and since then the patient has not been able to get upstairs without resting, because of pains in the bones; when the pains were bad the veins swelled. The catamenia began at eighteen, and since then have been regular monthly, lasting about five days, moderate in quantity; there has been no special pain, but the general shooting pains have been worse at the periods.

Mrs. A— was married December 26th, 1887; at that time she was straight-backed, and her height then and for a year afterwards was 5 feet $1\frac{1}{2}$ inches. The present height, ascertained by measurement, is 4 feet $7\frac{1}{2}$ inches, so that the patient has lost six inches since 1889.

The first child was born in October, 1888, and was suckled for two months; during this pregnancy the woman says she was well except for "falling of the womb." The labour was natural.

The second child was born in November, 1889, and suckled for two months; during this pregnancy there was "falling of the womb" all through, and much neuralgic pain about the abdomen. On getting up the patient was bent forward, and could not straighten her back. About a month after the confinement the left knee became swollen, and remained so until the third baby was born. The onset of the swelling was accompanied by much pain in the joint, which, however, soon disappeared. About the same time there was creaking on movement of both hips and both knees; the general strength failed, but she did not become shorter until the third pregnancy.

In October, 1890, she was in the General Hospital under the care of Dr. Wade, when it was noted that the shooting pains had been much worse in the preceding twelve months; at this time the pains, which had

formerly been seldom and of short duration, lasted with short intermissions for hours and even days; they were worse in the legs, and would cause the patient to fall down if standing; when severe, they were followed in about an hour by a pain in the cardiac region, causing a fluttering of the heart. About three months before this, that is in July, 1890, patient had the "dropsy;" the water gathered in about six days, and the doctor is said to have removed six pails of water from her by tapping; the patient did not keep her bed, and there has been no return of the dropsy. While under Dr. Wade it was noted that the woman was five months pregnant. The chief points at this time in the condition of the nervous system were slight anæsthesia in both legs, loss of knee-jerks, slight nystagmus, complaint of dulness of vision, difficulty in standing and walking, and severe pains. There had been tired, aching pain, and loss of use of the right leg for a fortnight; the same leg always felt cold and numb above the knee; it swelled at night; even when thoroughly warmed at the fire, the former numbness quickly returned. There was swelling about the right great trochanter, and when the patient stood on the right leg there was marked lordosis. A lateral curve of the upper dorsal spine was also noted at this time. The pupils were observed to react both to light and accommodation.

This third pregnancy terminated in natural delivery in March, 1891; a month before the confinement the patient had a severe fit; there was a second severe one at the end of 1891, and since then there have been many slight ones in which speech and sight, and sometimes consciousness, have been lost; the mouth worked, but the mother of the patient used to prevent tongue-biting with a spoon placed between the teeth; urine has never been passed in an attack.

In July, 1891, the woman was admitted under the care of Mr. Barling with tabetic arthropathy of the right hip-joint; the limb had gradually shortened, and was now

two and a quarter inches shorter than the left one; the swelling of the left knee was much less, but there was still some thickening around the margin of the tibia and femur. There was a small, freely moveable piece of bone connected with the right anterior superior iliac spine. Sight had improved, there was still slight nystagmus. The lameness had increased, and the gait was peculiar and characteristic.

Shortly after leaving the hospital in September, 1891, the woman says a raised boot was made for the right foot, the leg having become four inches shorter than the left: but before she could wear it the left leg suddenly, without any pain, became as short as the right.

In 1892, there was difficulty in telling the position of the feet, the patient not being able to tell, for instance, whether they were in bed or out of it, without looking at them. In the same year she was cross-eyed for about three months.

The fourth child was born in September, 1893, and was suckled for about a month; during this pregnancy the patient did not feel at all worse. There have been two monthly periods since this labour, the last ending on January 2nd last. For nine months there has been occasional difficulty in holding and in passing urine. The bowels have varied, sometimes being very stubborn, and sometimes acting regularly; in the last six months they have on a few occasions moved without the patient's knowledge.

The woman has observed no difference in the character of the pains in her various confinements, which have all been natural and easy; the third and fourth were, if anything, easier than the first two; the fourth was over before the doctor could reach the house; there have never been any after-pains.

The second child died when ten months old of "diarrhoea and dysentery"; the third at two years of "inflammation of the lungs, bowels, and brain." The first and fourth are both living and well; the first one is a

well-developed five-year-old of quite healthy appearance, with good physiognomy and teeth.

Since marriage the patient has always lived in a dry and comfortable house, but has had at times difficulty in getting sufficient nourishment. Before marriage, and for some time afterwards, she worked at rule-making.

Family history.—Father and mother alive and well, except that the latter has a patch of lupus affecting the right side of the nose, right eyelids, and cheek; this has existed for twenty years, and is still active. The father's sister had her backbone curved from childhood, was never able to speak or feed herself, and died at twenty-three. Two maternal uncles died at thirty-three and forty-four respectively.

The patient is the fourth of eighteen children, of whom the first six and the last one grew up; all the others died at from nine to eighteen months, many of them in convulsions. The eldest brother is "afflicted"; notes of an examination of him are appended to this paper (since the notes were taken, the man has on one occasion tried to commit suicide by hanging himself). The third brother committed suicide. The second, fifth, and sixth, and the youngest of the family, three males and one female, are living, and are said to be healthy and strong.

Present state.—Mrs. A— is 4 feet 7½ inches in height, of fair general muscular development, but with little subcutaneous fat. A scar on the front of the left thigh is said to be the result of a burn at the age of eight.

The patient can stand without support, but has to hold on to something to steady herself in walking; the chief difficulty in walking is at the hip-joint (Plates I and II).

While standing, there is a deep hollow over the lower lumbar and upper sacral regions; the increased curve, with its concavity looking upwards and backwards, affects the whole of the lumbar and the upper sacral vertebræ. There is also some lateral curvature of the dorsal spine, with the convexity to the right. Viewed laterally, the buttocks project about five inches behind the



Adlard, imp.









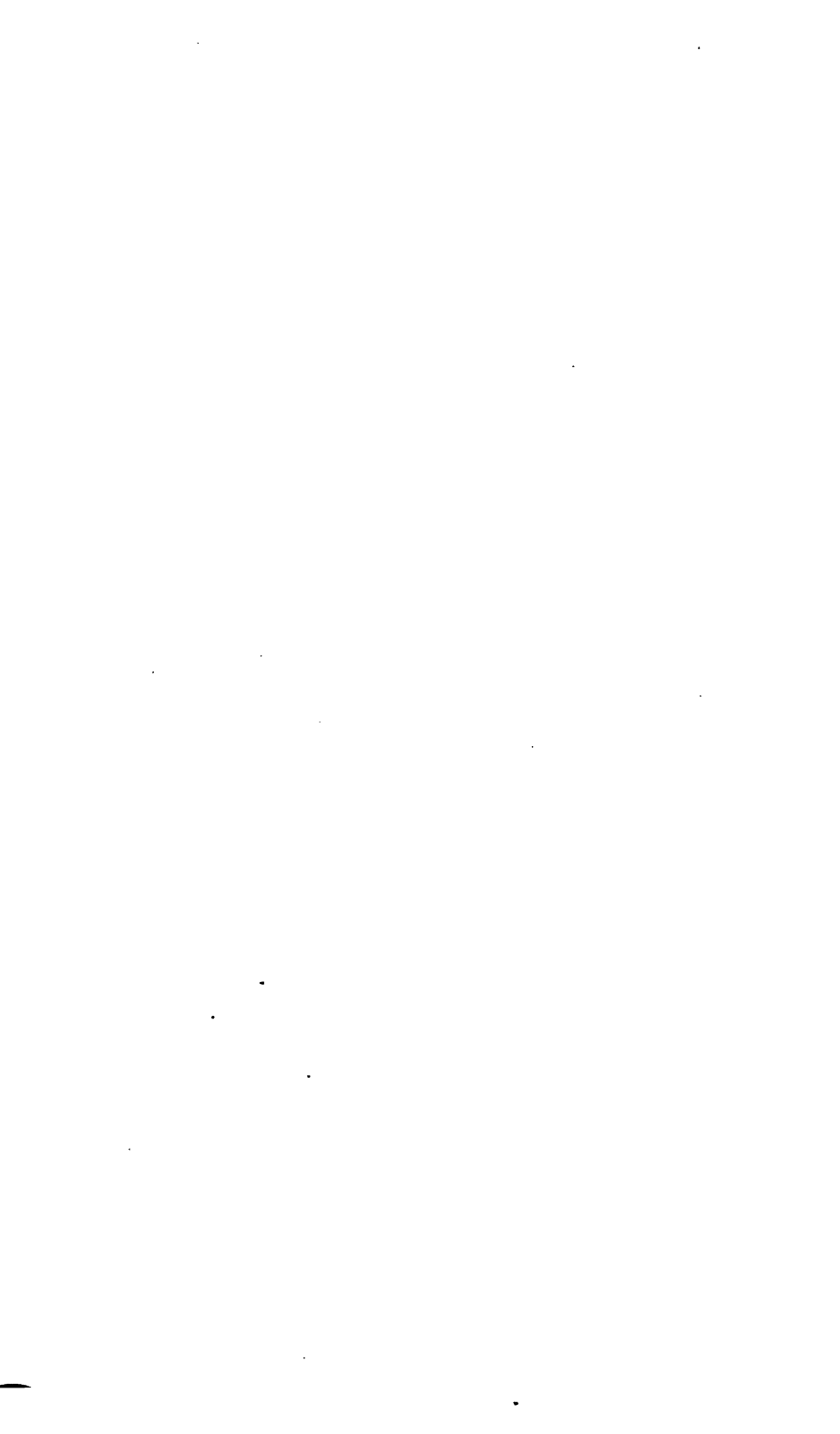
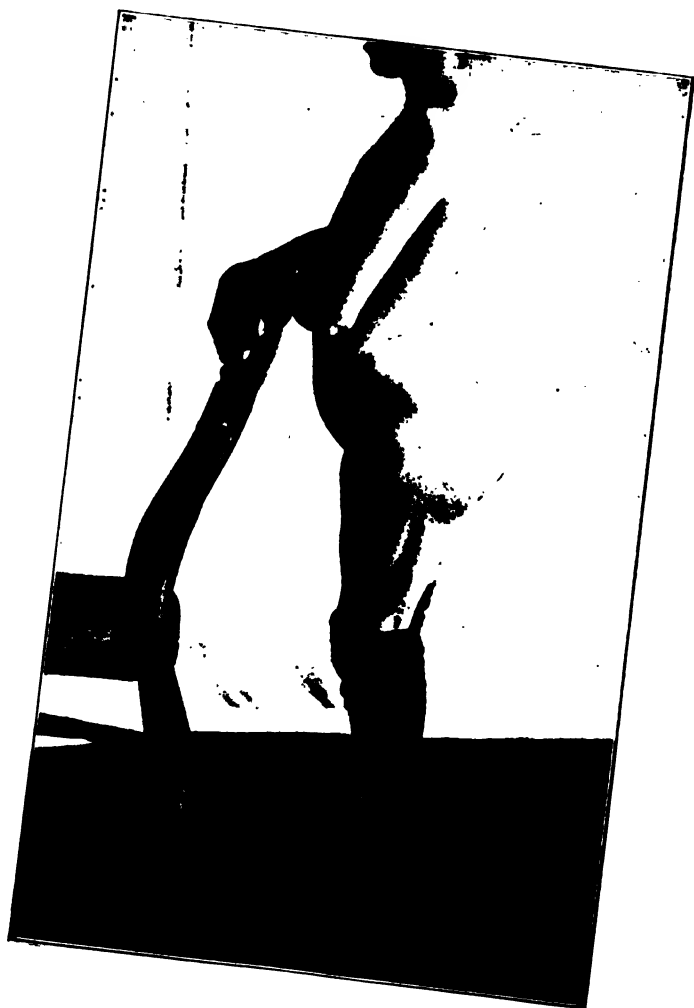


PLATE IV.

'Med.-Chir. Trans.,' Vol. LXXX.



Adlard, imp.



vertical level of the shoulders, and six inches behind the upper lumbar spine. The abdominal wall sweeps downwards and backwards in its lower half, forming almost a semicircle from the epigastrium to the pubes, which latter is displaced backwards. The iliac crests run downwards and forwards from the posterior superior spines, and their anterior two thirds are nearly vertical (Plate III).

The right anterior superior iliac spine presents a small depression between two prominent points of bone, as if a small piece had been broken off. The great trochanters form prominent projections, extending upwards on either side to within an inch of the level of the highest part of the iliac crest, and an inch higher than the posterior surface of the upper part of the sacrum, which is horizontal (Plate IV).

When the woman sits down or lies on her back, the lordosis above described completely disappears. As the patient lies on her back, the length of the legs is measured from the anterior superior spine to the tip of the internal malleolus. The right leg measures $29\frac{1}{2}$ inches, and is increased by a moderately strong pull to 31 inches; the left one is 30 inches at rest, and 31 inches when extended.

Joints.—There is much grating in both hip-joints, and passive mobility is free, the legs being able to be lengthened by one or two inches, but immediately becoming shortened again on being left free; both grating and mobility are better marked in the right joint. There is no pain on manipulation. Both knee-joints are distinctly loose, and allow a small amount of abnormal mobility; this is more marked in the left. Nothing abnormal is observed in the ankles, and there is no deformity of the chest or upper limbs.

When the patient stands lightly touching the back of a chair, some jerky, irregular movements are observed affecting the arms and trunk. There is marked unsteadiness on standing with the eyes shut.

Lying on the back, the woman cannot raise her legs from

the couch, and when the legs are crossed she has to lift the upper one with her hands in order to get them uncrossed. The knees can be raised fairly well, the feet remaining on the couch. All the movements of both hips, except abduction, are fairly performed; abduction is slight, being interfered with by the position of the upper ends of the femora. The movements at both knees and ankles, and of the joints of both feet, are all well performed.

There is no special wasting of the lower limbs; no rigidity of muscles.

The knee-jerks are both absent; there is no ankle-clonus.

Sensation.—There is deficiency in both tactile and painful sensation in both feet, particularly over the soles. Thermal sensation is tested with glass tubes containing hot and cold water; above the knees these are correctly discriminated, but the patient is usually wrong in her answers about touches with them on the skin below the level of the knees. When the eyes are closed, and the legs passively moved about, and the woman is then asked to describe the position in which the limb is placed, she is always mistaken; this statement applies to both legs, and to the position of knees, ankles, feet, and toes.

The feet are often cold, but sometimes hot and burning. There are severe pains in various parts of the body, sometimes occurring on two or three days in one week, sometimes not for two or three weeks; they are less frequent than formerly. The most common situation is in the arms, beginning about the shoulders and running down to the fingers; the arms "go dead" until the pains are gone, the woman being unable to raise them. The head is also frequently affected, always on the right side of the vertex and above the right ear. Less often now than formerly, pain occurs down the legs along the front of the bones and in the calves. Occasionally there are lower abdominal and inguinal pains.

There is no plantar reflex on either side; the abdominal

and epigastric reflexes are present, more marked on the right side. Sexual desire has never been excessive; for the last few months both desire and gratification have been somewhat diminished. There has been no difficulty in micturition since the bladder was emptied by catheter and the uterus reduced into the abdominal cavity.

Examination of the face, tongue, arms, and hands, reveals no abnormal physical signs. The pupils are below the medium size, do not react to light, contract sluggishly with accommodation, do not dilate on pinching the skin of the neck. The movements of the eyeballs are good in all directions. The ophthalmoscope reveals nothing abnormal. The corneæ are clear. The teeth are good; particularly the incisors are well formed. The patient displays more than the average intelligence of women in her station in life; her memory is very good.

Genito-urinary.—The catamenia have been absent since January 2nd. The bladder can now be emptied voluntarily. The uterus, which is of about the size usual at the third month of pregnancy, is now entirely above the pelvic brim, and is felt in the lower abdomen to the right of the median line, and extending upwards as high as the umbilicus. *Per vaginam* the os is felt high up, the cervix partly softened. The sacrum appears to have its normal curve, and there is no lateral contraction of the pelvic cavity.

Inclination of the pelvic brim.—When the patient lies on the left side, the line joining the promontory of the sacrum with the upper border of the pubic symphysis appears to form an angle of about 10° *in front of* the long axis of the body. On standing, the inclination of the brim is estimated by placing the tip of the finger on the promontory and the thumb on the top of the symphysis; the line joining these two points extends downwards and a little backwards, making an angle of nearly 10° *behind* the vertical axis of the body.

Measurements of the pelvis.—The diagonal conjugate diameter is easily measured by two fingers, and the

average of several trials gives $4\frac{1}{2}$ inches. Dist. sp. il., 10 inches; dist. cr. il., 11 inches; dist. tub. isch., 4 inches.

Examination of the cardiac, respiratory, and renal systems discloses no abnormal physical signs.

Progress of the case.—On April 3rd the patient reported that there had been some dirty-reddish discharge for four or five days; the cervix was somewhat patulous. The red discharge continued, and on May 13th miscarriage took place. I am indebted to Dr. J. J. Kerr for the information that the woman was delivered of a five months' foetus with membranes and placenta intact, after having strong labour pains for two days; the delivery was natural, and recovery took place naturally.

On July 17th I again saw Mrs. A—, and found her general appearance decidedly improved. The uterus was of natural size and mobility, there was nothing abnormal disclosed on pelvic examination. The condition of the nervous system was carefully gone over, and found to agree in every detail with the notes previously taken and above recorded.

The patient was next seen on February 26th, 1895, when she was found to be again nearly two months pregnant. After this visit she was lost sight of, and was not found until February 28th, 1896, when she said that she had had to go into the Workhouse Infirmary to be confined. The delivery took place on September 18th; for a fortnight previously there were "forcing" pains, which continued much the same in character till the child was born; forceps were applied. There was no trouble with the after-birth. No difference was noticed by the patient in the character of the pains of the second stage between this and former labours. The child is living, strong, and well; it was suckled for a time, but the mother's milk began to diminish at three weeks, and finally disappeared at the end of two months.

For two months before the confinement the woman says she could not stand or walk, nor even sit up; on several occasions she fell off a chair. The inability to

stand or walk continued for two months after the labour, and then rapidly disappeared.

On this date, February 28th, 1896, the condition of the patient was again carefully gone over. The pains seemed to have become, on the whole, less marked and severe than they were. The ability to walk and stand, and the power of movement in the legs, were the same as before. Sensation of all kinds was about the same, except that there was now deferred sensation of the prick of a pin on the soles of both feet. Micturition is normal, except that a call has to be promptly obeyed; frequently a motion is passed unawares, on these occasions there is no warning sensation, and if there were the patient does not think she could control the action. Sexual desire and power are about as they have always been. The pupils are small, 2.5 mm., equal; reactions as before; optic discs normal. Memory and intelligence are perfectly good.

Locomotor ataxy is said by Dr. Gowers, in his 'Manual of the Diseases of the Nervous System,' to occur ten times more frequently in the male than in the female sex; and Erb in one series collected 350 male to only nineteen female cases of the disease. Marie is of opinion that probably 4 to 5 per cent. of cases of tabes suffer from arthropathy, and the joint affection might therefore be expected to be very rare in women. This, however, does not appear to be the case, for in a number of cases of tabetic arthropathy Weizsäcker found seventy-two males and thirty-nine females; although this probably represents too high a proportion of females, it seems to indicate that among patients affected with tabes, females are more liable than males to have the joints affected.

Tabes rarely begins below the age of twenty, and not very commonly under thirty. In the case under consideration the disease began with lightning pains in the lower limbs at the age of sixteen, and the joint affection began at twenty-four during the second pregnancy, dislocation of both hips occurring at the age of twenty-six.

Since this time two pregnancies have gone on to term, and one has ended in miscarriage at the fifth month. Such a case is certainly very rare, and I have been unable to meet with the record of a similar one.

The clinical picture is unusually full of detail: unsteadiness on standing with the eyes closed, athetoid movements, loss of knee-jerks, affection of tactile, painful, and thermal sensation, loss of muscular sense, spontaneous fracture of the ilium, disease of both knees and both hip-joints, temporary strabismus, Argyll-Robertson pupils, pains of various kinds, paræsthesiæ, and some affection of the sphincters,—all were observed.

As regards the ætiology, acquired syphilis, which is a factor in the previous history of from 58 to 88 per cent. of patients suffering from tabes, can, I think, be excluded from the present case with confidence. There are, besides, no signs of congenital syphilis either in the patient or in the brother who was examined. On the other hand, there is a strong family predisposition to nerve degeneration, as evidenced by the paralysis in a paternal aunt, the suicide of one brother, and the attempted suicide of a second.

In four other cases of tabes in females of which I have notes, in two there was a strong presumptive evidence of acquired syphilis; in one, the ataxic symptoms began at twenty-one, and between the ages of sixteen and thirty-five there were nine miscarriages, all at the third or fourth month: in the second there were three children stillborn at term, and one miscarriage at the third month; a sister of the patient suffered from fits.

No clear effect on the progress of the nervous affection in Mrs. A— can be attributed to the repeated pregnancies. The symptoms of tabes seem to have increased rather rapidly from the time of the second delivery onwards for about two years; after this they again became stationary. No material difference in the symptoms and signs has been detected by careful and repeated examinations in the two years during which the patient has been under my observation.

From the obstetric point of view the case is interesting as showing the effect of simple increased inclination of the pelvic brim on pregnancy and labour. The lordosis, and with it the increased inclination, began in 1889, when the woman was twenty-four years of age; the pelvis would then have quite or nearly reached its full development. At the present time, taking the promontory of the sacrum as a centre, the plane of the pelvic brim has rotated downwards through an angle of somewhere about 40° , so that in the erect position the plane of the brim looks forwards and a little downwards. Measurement shows that we have to do with a flattened pelvis of average transverse size and development; the true conjugate is about half an inch below the average. This flattening has given rise to no difficulty which has called for interference during delivery until the last occasion, when labour was completed by forceps.

The rotation of the brim is due in the first place to the lordosis secondary to dislocation of the hips, and in the second to rotation backwards of the anterior or lower half of the os innominatum by the ilio-femoral ligaments, and by the action of the muscles which are attached by one extremity to the ischium and pubes, and by the other to the upper part of the femur. The muscles which will have the greatest effect are the internal and external obturators, the quadratus femoris, the gemelli, and the upper part of the adductors, especially the adductor brevis. The pectineus, the piriformis, and the psoas and iliacus, will aid the rotation to a less extent.

The lordosis, and with it the increased inclination of the pelvic brim, disappears when the patient sits or lies down; this is a proof that the increased curve is not due to weakness of the spinal muscles, but to the dislocation of the hip-joints, and the consequent lessening of the effect of the extensors of those joints. It has been pointed out by Dr. Gowers that the lordosis which is frequently met with as an early symptom in pseudo-hypertrophic paralysis also disappears on sitting, and is thus proved to

depend on weakness of the extensors of the hip and not of the trunk muscles.

The influence of the alteration in the pelvic inclination was shown in the fifth pregnancy by the uterus becoming incarcerated in the pelvis at the tenth week ; this was the state of affairs when the patient first came under my observation. In the sixth pregnancy the womb rose into the abdominal cavity without any trouble. After entering the abdomen the uterus lay entirely above the brim, so that at the third month it already extended as high as the umbilicus, a height which under ordinary circumstances it only attains at the fifth month. The uterus was displaced entirely to the right of the middle line by the prominence of the vertebral column.

On labour the alteration in the pelvis seems to have had no appreciable effect. The increased inclination of the brim would tend to prevent the head entering ; it was observed, however, that the lordosis, and with it the alteration in the inclination of the pelvis, disappeared when the woman assumed the recumbent posture.

On the pains of labour, the disease of the nervous system, in spite of the impairment of sensibility associated with it, appears to have had no influence. Tabes does not, as is well known, necessarily affect the visceral nervous system, although it may do so ; as Dr. Gowers has pointed out, a fatal pleurisy may be painless.

My thanks are due to Sir Willoughby Wade and to Mr. Gilbert Barling for allowing me to make use of the notes taken when the patient was under their care in hospital ; and I have also to express my sense of obligation to Mr. Bower, late house surgeon at the General Hospital, Birmingham, for his kindness in photographing the patient.

APPENDIX.

G. D—, the eldest brother of Mrs. A—, was seen and examined on March 18th, 1895. His age is thirty-four, height a little over 5 feet; appearance that of a partial idiot.

Speech is very defective, the man being often unable to find words to express himself, and pronouncing those he uses very thickly and imperfectly. He seems to understand well what is said to him in the course of the examination, obeys orders intelligently, and, in fact, investigation shows that he is possessed of a much higher intelligence than would at first sight be supposed. He is said to be able without inconvenience to do without sleep for three or four nights at a stretch, and he often sits awake by the fire all night. His appetite is said to be very large.

The face is decidedly lacking in expression, smooth, the natural furrows few and badly marked. The action of all the muscles on both sides of the face is very defective. Frowning is done slightly, but very imperfectly; the forehead cannot be raised as in surprise; the man cannot grin to order, the left angle of the mouth moving only slightly, the right not at all; both angles move when the patient is made to laugh; the patient can whistle. The tongue is protruded a little to the right. The eyes move fairly in all directions; there is no nystagmus; the pupils react briskly both to light and accommodation. The ophthalmoscope shows nothing abnormal. The upper limbs are normal in power and movements. No radius or triceps jerk can be obtained on either side. In walking the man projects his body somewhat forward, keeps his legs bent at the knees and the toes pointed, and progresses with short quick steps. The leg muscles are well developed; they offer considerable involuntary resistance to passive movements. The knee-jerks are very active, equal on the two sides. There are well-marked clonic movements at the ankles, while the feet retain a partly extended position against pressure, but there is no true ankle-clonus. There

is no defect in tactile or painful sensation anywhere in the body. The plantar reflexes are very active, equal on the two sides. The cremasteric, abdominal, and epigastric reflexes are equal. Sometimes, perhaps once in two or three days, there is involuntary micturition. The bowels are regular, and never act involuntarily.

The condition above described dates from infancy, and is said to have come on with "fits." The brother-in-law who accompanies the man says the disease has been at a standstill ever since he has known him, that is for at least ten years. The patient earns some money by selling papers in the street, and seems to be fairly good at this particular line of business.

Since these notes were taken the man has on one occasion tried to hang himself.

The affection of the nervous system in this case seems to be most likely due to meningeal hæmorrhage from injury during birth, resulting in degeneration of certain of the convolutions, notably those having to do with the movements of the face, tongue, speech, and legs.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 9.)

ON A CONDITION OF MIXED PREMATURE AND IMMATURE DEVELOPMENT

BY

HASTINGS GILFORD, F.R.C.S.ENG.

(COMMUNICATED BY MR. JONATHAN HUTCHINSON)

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IN the 'Transactions' of the Royal Medical and Chirurgical Society for 1886 is a paper by Mr. Jonathan Hutchinson, entitled "Congenital Absence of Hair and Mammary Glands, with Atrophic Condition of the Skin and its Appendages, in a Boy."¹ Last year I brought to Mr. Hutchinson's notice a case in which a similar condition obtained in a youth of seventeen, who had been under my observation for nearly four years.

My patient died on December 15th, 1894, and as I had the good fortune to obtain a series of photographs of him at different ages, and was permitted to make an examination of the body after death, I am able to give a fairly complete history of his condition from beginning to end. Mr. Hutchinson has already written a short

¹ 'Med.-Chir. Trans.,' vol. lxi, p. 36.

description of this case in his 'Archives' for April, 1895. I am indebted to Mr. Hutchinson, not only for the light he has there thrown on the clinical features of the case, but for his permission to complete my account by the description of the present state of the patient he saw nearly twelve years ago. I have also to thank Dr. Jago, of Plymouth, for the very cordial way in which he both met my request to see his patient and helped me in my investigation of the case. I propose now to give a detailed description of my own case, and afterwards to add a short supplementary report of the present condition of Mr. Hutchinson's original case.

CASE 1.—*Family history.*—The grandfathers on both sides were subject to gout. The father and mother are well developed, and a little above the average height. There is no history of insanity, consumption, rheumatism, acromegaly, or syphilis. Of the four brothers and five sisters, all have been sound and well formed from birth, except one girl, who had rickets when a baby; she is now, however, in perfect health, and is without a trace of deformity.

A. R.—was the fifth child. There is not the slightest facial resemblance between him and the other members of his family, with one exception. I was shown the photograph of his paternal grandfather, taken when he was over seventy, and there certainly was in this instance a likeness between the two. But it was the likeness of one old face to another rather than that of two members of the same family. The resemblance lay in the shrivelled skin drawn over the wasted soft tissues and protuberant bone, in the aquiline nose, thin lips, white hair, and the general aspect of repose of old age.

Personal history.—His mother remarked no unusual circumstances while carrying him, except that she was nearly always "unwell." He was born at full time, and was a small and fretful child, but was in no other way remarkable during early infancy. He was never

of a bluish colour. His hair and nails were well grown, and though not fat, he was by no means emaciated. His mother attributes the commencement of the disease to teething. He began to cut his teeth when about six months old, and from that time his hair began to fall off, his nails to shrivel, and his fat to diminish. At the age of eighteen months, when his first photograph was taken, more than half his hair had thinned off, that running along the middle of the vertex of the head persisting longer than the rest. The shoulders were rounded, the chest narrow, and the cranium large in proportion to the face. The dropping of the head and consequent upward direction of the eyes, which in the photograph give a hydrocephalic look to the face, were said to be a characteristic feature at this period. But in the next likeness, which was taken at the age of seven, this furtive expression is quite absent, and though the hair is there as scanty as it was when I saw him, his hands and face look by no means devoid of fat. Indeed, the cheeks yet show evidence of the presence of the sucking pads of early childhood. He was a lively, good-tempered child, but was so easily fatigued that he could never run about with other children.

At the time of the third photograph (at the age of twelve) the face had assumed the sedate expression that it has in the last photograph of all, and the whole type of face and figure is that rather of an adult than of a child. His mother says these photographs are truthful representations of his appearance at these ages.

At the age of eighteen months he had an offensive mattery discharge from the nose, and afterwards from the left ear, which did not quite disappear until he was fifteen. He never snuffled in infancy, nor did he have rashes or sores round the mouth or on the bottom or cheeks. He was never double-jointed, nor did he at this time sweat unduly, and his limbs were always straight. He did not walk until nearly two years old, and was backward in learning to speak. Though he

began to cut his teeth at the age of six months, the process of dentition was very slow, and it is not known when the second dentition commenced. He had measles when about four years old, and at eleven became dangerously ill with broncho-pneumonia, from which he did not recover for seven weeks. He also suffered from attacks of flatulent dyspepsia from "as early as could be remembered." These were attended with epigastric pain, and occasionally with nausea or vomiting, and they became more frequent and persistent towards the end of his life.

From the time of the appearance of the first indication of his disease his breathing was difficult at times, and he could never hurry because he was so "short-winded." He told me that occasionally he could "hardly get his breath," but I myself never saw him in this condition. His mother confirmed his statement, but said the attacks never caused him to change colour, but were evidenced by the quickness of his breathing and by its asthmatic character. In the night he always lay with his mouth open, and his breathing was then sometimes so uneasy, and the noises he made so uncouth, that those who were with him feared he would be suffocated in his sleep.

Condition when first seen.—A. R.— was first brought to me in January, 1891, when he was a little over fourteen years old, but his appearance as he came into my room was suggestive either of a child of five or of a wizened and dwarfish old man. On the one hand, his size (height 1.04 m., weight 16.34 kilos.), manner of dress, and the fact that he was brought by his sister in a mail-cart, drove one at once to the conclusion that he was a child. On the other hand, had he come by himself in the clothes and other accessories of old age, I think there are few who would have detected the deception. This appearance of senility was occasioned not only by his baldness and by the wrinkled condition of his skin, but by the leanness of his figure and by the lack of that vivacity of carriage and expression which is one of the chief features of early

life. The thighs, moreover, were so thin as to give an appearance of undue width of the fork, and this, together with a slight stoop from the hips, a want of fulness in the buttocks, and a perceptible stiffness in gait, still further increased his resemblance to a decrepid old man. He was, however, neither tremulous nor tottering in his walk. The veins of the backs of the extremities, and of the neck, forehead, and scalp were large and very conspicuous, those in the last situation being readily seen from a distance of more than one hundred feet. They were asymmetrical, and consisted principally of a very large left temporal, which inosculated freely with a right frontal vein and with a few smaller branches of the right occipital. But what contributed more than all to his "old-mannish" appearance was the withered and juiceless look of his skin, the absence of colour in the scanty hairs on his scalp, and the piping tone of his voice. The eyelids and eyebrows, too, at first sight appeared devoid of hair, but on close inspection a few scattered and downy hairs were seen in both situations. A few were also to be seen on the backs of the hands and wrists, but on no other part of the body. The outlines of the cartilages of the nose could be seen with startling distinctness. They were slightly but unmistakably hypertrophied, so as to give an aquiline turn to that feature quite different from the shape of the nose of other members of the family. The cartilages of the ears were also large and well formed, but the lobules were absent. The nails of the fingers and toes were flat, ill-shaped, and membranous, and as short as though they had been bitten to the quick, though no nail-bed was visible. There was no vestige of mammary glands, and the nipples were unusually small. The umbilicus was a mere indentation in the skin, without folds. The skin was thin, soft, and pliable, and, though usually dry when I saw him, this was probably due to his very quiet habits, for on two occasions in hot weather I was able to detect the presence of sweat of normal reaction. He himself, and his mother, said that he often

sweated profusely. The colour of the skin was slightly brownish, with a tinge of red over the trunk, especially in front, where it was sprinkled thickly over with small non-pigmented spots as if he had been sprinkled with rain. This peculiarity was visible only on near inspection, except at the back and sides of the neck, where the colour was more pronounced. Tactile sensibility and the perception of heat and cold, as well as the senses of taste and smell, were fairly acute. He always felt extremes of heat and cold very severely, and on cold evenings in the winter was always carried up to bed wrapped in blankets. But while his body and limbs were cold, his head was generally hot, and even on cold days he would sometimes bathe it in cold water to relieve the feeling of heat. His temperature was always nearly normal when taken. Both superficial and deep reflexes were absent.

With the right ear he could hear the tick of a watch at ten inches, though the drum was represented by a mere ring. The only ear bone that could be seen was the incus, which was very large, and was exposed for nearly the whole of its extent. It should, however, be remembered that the ear bones do not grow after birth, and it was probably therefore only relatively large. On the left side there was a small perforation at the hinder and lower part of the drum. The membrane was tense, and the handle of the malleus very prominent. A watch could be heard on this side at a distance of twenty-one inches. The left Eustachian tube was patent, but no air could be got through the right.

The eyes seemed large and protuberant; but on comparison with those of another youth it was evident that this appearance was partly due to the narrowness of the palpebral aperture on the one hand, and the absence of surrounding fat on the other. There was no arcus senilis. The left eye showed an expansion of the semi-lunar fold into an imperfect nictitating membrane, which overlapped the cornea for about 3 mm. There was hypermetropia of both eyes, with slight astigmatism, which I

found was best suited with glasses of + 4·5 D. for the right eye, and + 2· D., with the addition of + ·5 D. in the vertical diameter, for the left eye. In other respects his eyes were normal.

His intelligence was uncommonly good, and though he was thrown much into the society of children his ideas were those of a man, and he could take his part in the small talk of adults with interest and intelligence. His memory was of average ability. He had had no schooling, but had taught himself to read easy sentences. He was quiet in manner and gentle in disposition, and his good nature and maturity of thought and judgment were shown in his consideration for others. Owing to his dread of rebuffs he was averse from the society of strangers, but was liked by those who knew him. When tired he often complained of headache, and then rested his head on his hands to relieve himself of its weight.

His muscles were not well developed, and were easily fatigued. He was unable to walk more than a mile at any time without feeling tired, but as a rule was drawn by his sister in a mail-cart.

In its more obvious aspects, his body was not badly proportioned ; for though his head was evidently large, it was not relatively disproportionate for one of his stature. For Sir G. M. Humphry¹ has pointed out that the heads of short people are naturally as large as, and may be even larger than the heads of those of ordinary height. In A. R—'s case, however, the head was actually slightly smaller (16·5 cm. long, 14 cm. broad, 49·4 cm. in circumference) than that of the average adult (54 cm. in circumference).

The length of the limbs, too, was for a youth of his age not disproportionate, for the middle point of the total length was less than an inch above the symphysis pubis.

But in other respects he was not only stunted in growth but deformed. In the first place the right half of the

¹ 'The Human Skeleton,' p. 96.

cranium was smaller than the left, the latter projecting nearly 2 cm. behind the former, but being level in front. The forehead was not overhanging, and the orbital plates of the frontal bone were horizontal. There were also no bosses on either of the parietal bones, and both they and the occipital showed no wasted areas. The anterior fontanelle was, however, open to the extent of about 5 mm., and the pulsations of the brain were easily seen. The margins were not thickened, and the lines of the sutures could not be distinguished. In the next place the shoulders were rounded, and the chest narrowed by the smallness of the clavicles, which were so puny as to remind one of the "merry thought" of a chicken. Each measured no more than 6 cm. in length. The scapulæ were also small.

Lastly, while the shafts of the long bones of the limbs were decidedly thin, the ends were relatively thick. Thus on comparing his hands and feet with those of another emaciated boy, it was manifest that not only were the knuckles conspicuous by the absence of fat, but they were also enlarged. Some epiphyses were large in proportion to others. This, as Mr. Hutchinson had pointed out, was the case with the lower ends of the humeri and the upper ends of the radii. But the lower epiphyses of the femora were still more disproportionate in size. While the limit of its articular surface projected well beyond the level of the edge of the articular surface of the tibia behind, in front the effect of the undue prominence of the condyles gave an appearance to the tibia of being partially dislocated backwards. It also gave rise to an exaggerated prominence of the large patella, and caused its ligament to be set into its tubercle at an angle of 135° with the shaft. It was perhaps, some corresponding disproportion in the size of its articular surfaces that caused the hip-joint to be stiff. He could not bend downwards sufficiently to lace his left boot, though he could just manage the right. I was unable to ascertain whether this explanation was correct, nor could I be sure whether

a similar cause accounted for his inability to fully open his mouth.

His lips were thin and compressed, and formed with the mouth a conspicuous exception to the senile type of his other features. Instead of falling in so as to give the well-known crab-claw aspect to this part of the face, the alveolar portion of both jaws was unusually prominent. The effect of this was as if he were wearing a set of artificial teeth too large for his mouth. It also gave to the face, to a slight extent, the shape of a hatchet. The jaw could not be opened widely enough to permit of an examination of the fauces. The frænum of the tongue was so short that he could hardly project it beyond the teeth. The tongue itself was small, and not so thick on the right side as on the left, and was smooth and almost glazed on its surface, owing apparently to a deficient development of papillæ. It was never furred. Both maxillæ were contracted, and the two sides of the palate were convex downwards from side to side so as to meet at an angle and form a furrow along the middle line. The teeth were few in number, and very irregular both in level and shape, though the sockets of the milk teeth were raised to the same level as those of the permanent. They were crowded and erratic in situation, and variable in size. Many were decayed, and some were not fully cut. The two upper central incisors, the molars, and the left first bicuspid were unusually large. They were arranged in two disorderly ranks, the upper teeth being set within the lower. There were ten in the upper jaw, of which two were left temporary molars, while the other eight belonged to the permanent set, six being greatly crowded canines and incisors and two right-sided bicuspids. The second bicuspid was much rotated and displaced. In the lower jaw there were thirteen teeth, of which six were much decayed milk teeth, viz. two central incisors, two last molar roots, one right anterior molar, and one left canine root. The roots of the lower milk incisors were abnormal in that they were little if at all absorbed

in spite of their successors being in place. Of the seven permanent teeth there were two greatly decayed anterior molars, three incisors, two canines, and one large bicuspid (on the left side).

His appetite was indifferent, and he ate little as a rule, preferring such soft foods as sweetbreads, milk puddings, and eggs; but this choice of diet was partly owing to the fact that his digestion was not good. He disliked fat, and when he forced himself to eat it, soon became "bilious" and had to give it up. He was also soon nauseated by cod-liver oil. His abdomen measured 45·8 cm. round at the level of the navel. The protuberance of the abdomen was probably in great part due to the size of the liver, which extended from the level of the seventh rib above to nearly halfway between the ribs and the navel below. The bowels were opened regularly every morning and evening.

No splenic dulness could be detected. His thyroid gland could be easily felt, and was in no way unusual either in shape, size, or consistency. There were no enlarged lymphatic glands, and no swelling could be felt at the root of the neck.

He soon became short-winded on exertion, though, as a rule, he respired through his nose at the rate of sixteen per minute in the sitting position. The chest measured 42·6 cm. round the line of the nipples on deep expiration, and 48·4 cm. on deep inspiration. On percussion over the chest, dulness was evident over a triangular area of which the base was situated about 1 cm. to the right of the sternum for nearly its whole length, while the apex was 2·2 cm. beneath and 1 cm. outside the left nipple. At this last spot a soft indistinct systolic murmur could be heard traceable into the axilla. At the base another bruit was distinguishable over the aorta, also systolic in rhythm, though less soft and of a more permanent character than that at the apex. The pulse was small, and varied in rate from 112 to 120 per minute. The temporal artery felt tortuous and thickened. On counting the number of red

discs in ten cells of a hæmocyto-meter, they were found to be of the usual size and shape, and to number 106·9 per cent. of the normal. A number of small granular masses were also present, but no nucleated red discs were to be seen. The white corpuscles were in the proportion of 1 to 346 red, and were of average size and appearance. The blood was examined on a warmed stage, and also diluted with neutral solution of sulphate of soda, and again with the acidified solution.

The rugæ of the scrotum were present, but were not so distinct as usual, giving the latter a smooth appearance at a little distance. The testicles were descended, and were of medium size. The usual sensation was experienced when they were squeezed. His urine varied greatly in character. Some specimens that he brought me were of average density, and some were pale and clear, and of a sp. gr. of from 1002 to 1006. The sp. gr. never exceeded 1025. The result of a careful observation continued for seven days in the spring of 1893 gave a daily mean of 396 c.c. in the twenty-four hours, a sp. gr. of 1019, and 12·6 grammes of urea. It was faintly acid, and contained neither albumen nor sugar. A specimen of sp. gr. 1008 allowed to stand for three months became dark in colour and aromatic, but did not decompose.

Subsequent history.—From the time that I first saw him in 1891, A. R— grew in height at the almost uniform rate of 2·5 cm. a year. The measurements of his head and abdomen were, however, always the same, while the girth of his chest increased by nearly 5·0 cm. The last measurement was made in October, 1894, when he was found to be 1·13 m. high. He seemed to age rapidly. His features became manifestly more shrivelled, and as he grew older he became still older in his ideas, and impatient of being treated as a child. When he had passed the age of sixteen there was evidence of sexual maturity. He occasionally had “wet dreams,” and on one of these occasions I was able to detect the presence of spermatozoa. The cartilages of the larynx, however, did not enlarge, and

there was no alteration in the peculiar tone of the voice. Neither was there any other sign of sexual maturity. He at this time began to suffer from rheumatic pains in his joints.

In the early part of the winter of 1893 I attended him during an attack of lobar pneumonia of the right base. The lung was consolidated from the base to an inch above the level of the lower angle of the scapula. His temperature at its highest was 103° . There was no sign of embarrassment of the heart, and notwithstanding great enlargement of the lymphatic glands of the neck the constitutional disturbance was slight. The crisis set in on the fourth day, and a gland over the left mylohyoid suppurating and breaking about five days afterwards, he speedily got well.

In the autumn of 1894 he became dispirited, more easily tired after exertion, more grave and subdued in manner, and less inclined for society. He was also more frequently ailing. His digestive trouble increased, and he sometimes complained of sharp pains in the left breast which shot down the arm. At the beginning of the winter he had a troublesome dry hacking cough.

On December 8th his last illness set in with diarrhoea and vomiting, and his usual pain and tenderness at the epigastrium. He was also unable to lie down on account of a choking sensation in his chest, which became worse when he was in the recumbent position. The breathing was slightly laboured and stridulous, though it was not distressingly difficult. No abnormal lung sounds could be heard. The pulse was 134, small and weak, and slightly irregular. No murmur could be heard at the cardiac apex, and the sounds were very indistinct. There was no cedema of the feet. The temperature was 101.4° . Liver dulness was increased, but there was no tenderness. The area of stomach resonance was manifestly extended, and he attributed much of his distress to "wind." Bismuth was given in half-drachm doses, with strophanthus and carminatives, and

these soon relieved the diarrhoea and sickness. The next day he was better, but on the morning of the third day he had a slight return of diarrhoea and was sick once, though his breathing was more natural. Later on, however, I received an urgent message to visit him, and when I arrived, found that while his mother was downstairs she had heard a noise from his room, and on running up had found him slightly livid, and sitting up in bed with his eyes and mouth open, trying to get breath. She took him in her lap, where he died quietly in a few minutes.

Post-mortem examination.—This was made two days after death. Rigor mortis was well marked.

On cutting through the skin it was at once evident that the subcutaneous fat was not entirely absent, though it was so scanty that it did not form a continuous layer. It was present over both the chest and abdomen, and was not thicker at the lower part of the abdomen. Fat was also present in still less amount in the mesentery and great omentum, where it was dotted here and there in the membrane, and was in meagre quantity behind the kidneys. There were no appendices epiploicæ.

The anterior fontanelle was closed, and the bone appeared to be of almost the same thickness there as elsewhere on the head, though pulsation had been evident less than three months before. The skull resembled in thickness and structure that of a child of a year old. It had an almost uniform thickness of about 2 mm., and there was no differentiation into compact bone and diploë. Neither the frontal sinuses nor the sella turcica were deepened, and the appearance of the base was otherwise normal. The ribs and their cartilages were of usual development, and the segments of the sternum were fully ossified, and all but the manubrium were united. The left clavicle was removed, and was with its cartilages 5.5 cm. in length.¹ It contained the beginning of a

¹ I find that the clavicle of a child of fifteen months old measures exactly 5.5 cm. without its cartilages. It should be remembered that "its mode of ossification is intermediate between that of a true cartilage bone and a membrane bone."

medullary canal. There was no centre of ossification at the sternal end, but the cartilaginous cap was very large.

The brain was normal in every respect. The convolutions were well formed and not flattened, and there was neither excess nor deficiency of fluid in the ventricles and subdural space. Neither the pineal nor the pituitary bodies were either larger or smaller than usual, and both were of healthy appearance.

On opening the thorax, a persistent and hypertrophied thymus came into view. Its lobes were nearly equal, and extended from about 2 cm. above the level of the manubrium to the junction of the fifth rib with the sternum below, overlapping the pericardium. They were of the usual shape, and when removed from the body the right lobe had a length of 7.2 cm., a breadth of 4.8 cm. at its widest, and a thickness of 2.3 cm., while the left was a little smaller. The two weighed 48.3 grammes together. The trachea was slightly flattened, but was far from being occluded by the pressure of the gland. The veins from the neck were not specially full. There was no ecchymosis. A microscopical examination was afterwards made, but owing to the thymus having been left too long exposed, no details of its structure could be made out, except a considerable increase in the amount of its fibrous tissue.

The thyroid gland was of relatively normal size and appearance, and microscopic examination revealed nothing amiss.

The lungs were quite healthy, but a few old pleuritic adhesions were present near the right base.

The heart weighed 121.0 grammes. There was no evidence of old pericarditis, and the muscle was firm and healthy. Both ventricles contained blood. The mitral orifice was not stenosed, but both valves were atheromatous. That half of the anterior valve which was attached to the ventricular wall was thickened with a large calcareous plate, but the free half of the valve was much less affected. The posterior valve was rolled up at its edge and incompetent. The bases of the aortic valves were studded with

cauliflower-like calcareous excrescences, almost surrounding the orifice. Above the right posterior valve was a large irregular patch, which extended for a height of 23 mm. up the aorta, and was composed principally of one unbroken plate continuous with that on the anterior mitral. Both coronary arteries were completely blocked, the right at its commencement and the left in the substance of the ventricle, where it felt like a bone in the muscular wall. Soft atheromatous patches occurred here and there over the whole of the aortic arch, especially along its convexity. On its concavity, opposite the origin of the left subclavian artery, was a large patch of calcareous material. Atheroma also extended into the branches, and a collection of organised fibrin almost blocked the origin of the right subclavian from the innominate artery. No trace of a ductus arteriosus was visible, and the coronary valve was quite closed. The tricuspid and pulmonary valves were healthy.

The spleen weighed 42·0 grammes; its capsule was thickened on its convex surface to the extent of from 1 to 2 mm., and was of cartilaginous hardness and consistence. On the concave surface the capsule was of little more than usual thickness. Its structure was normal.

The kidneys together weighed 130·0 grammes, and were neither hard nor congested. The capsules were slightly adherent. The supra-renal bodies were both of perfectly healthy appearance. The microscopical appearance of both kidneys and capsules was like that which occurs in the fibrous organs of the aged.

The stomach was dilated, and its mucous membrane markedly congested. The muscular and epithelial elements of both stomach and intestine were thin, and Peyer's patches were so atrophied as to be distinguished with difficulty. The liver was not congested: its structure appeared to be perfectly healthy; its edge extended to about halfway between the umbilicus and the edge of the ribs.

I was unable to weigh the brain and liver, and in some

other respects the examination had to be curtailed in deference to the wishes of the friends.

CASE 2.—The descriptive part of Mr. Hutchinson's original paper on this case is as follows, viz.:

"Congenital absence of hair and mammary glands with atrophic condition of the skin and its appendages in a boy whose mother had been almost wholly bald from alopecia areata from the age of six."

"The subject of this case, a boy aged $3\frac{1}{2}$, presented a very peculiar withered or old-mannish look, all his features being thin and pinched. His fingers were shrivelled and dusky, and their nails, which also were remarkably thin, were curved backwards so as to present more or less of hollow in the middle. His head was large and the anterior fontanelle not quite closed; the scalp was exceedingly thin, and with the exception of a quantity of down, was quite bald. It looked semi-transparent and tight, and the veins coursing in it were everywhere conspicuous. The veins were probably larger than natural. A large trunk came down the forehead on each side of the eyebrows and communicated by a transverse branch at the root of the nose. The inosculations across the middle line of the scalp were many. There was a peculiar blue tinge about the lips; it involved the skin, and not the prolabium only. At first I thought that this was due to accidental staining, but after he had been half an hour in my room it much diminished, as did also the turgescence of the veins of his scalp. His lips were exceedingly thin. His teeth were all cut and were tolerably regular; but his incisors did not stand quite straight, most of them had some slight inclination into the mouth. On his shoulders he was so thin that his coracoids and the outlines of his acromion processes could be easily seen, the skin over them being not much thicker than brown paper. The tightness of skin was nowhere very conspicuous excepting on the scalp; thus, on the abdomen, arms, and thighs the integument was

quite loose, but everywhere very thin. His muscular development was slight in all parts excepting the thighs, which felt hard and had muscles quite out of proportion to the rest of his body (this remark does not apply to the buttocks). His genitals presented a very remarkable contrast to the rest of his body. The parts above the pubes and upper part of the scrotum were so full and plump that a suggestion occurred that he must have double hernia. This, however, was not borne out by examination, and I believe the simple fact was that the scrotum and adjacent parts of the skin were in the state of those of a normally stout child, while everywhere else the skin, subcutaneous cellular tissue, and panniculus adiposus were almost absent. The true scrotum was small, naturally corrugated, and occupied only the lowest part of the genital pouch which I have described. I do not think that there was anything very unusual in this state in a child, but must admit that possibly there was some excess of subcutaneous development about the pubes and root of penis. His testes were well placed and of normal size. His penis, except that there was phimosis, was quite natural. His toes and their nails were in the same condition as his fingers. He did not walk quite perfectly, always keeping his knees a little bent, but I could not make out any definite muscular defect. One other remarkable feature remains to be mentioned: he had no nipples, and their sites were occupied by little patches of scar. These scars were exceedingly superficial and slightly marked, but I am sure that they were there. Nothing like a mammary gland could be traced.

"The history which the mother gave me of the child was that he had had no ailments since his birth, was of cheerful disposition, and very intelligent. It had been necessary from cross-presentation to turn during delivery, and for some days after birth he had been very blue, probably in a state of partial cyanosis. He was still liable to vary very much in blueness in connection with

the temperature and states of excitement, but never now presented anything approaching a cyanotic condition."

Dr. Jago described his condition when he was fourteen years old in the following letter to Mr. Hutchinson:¹

"Plymouth, July 5th, 1894.

"DEAR SIR,—I am pleased to reply to your letter received this morning. Master B— was fourteen years old in April last, is 43 inches in height, and I guess his weight to be about 55 or 60 lbs. He presents the old man—even aged man—in his countenance. His voice is fairly good, but has a very peculiar piping sound. There is no want of intellect, and his fondness for study is more than usual among lads of his age. His general health is good, and he is very active. When an infant he had cyanosis, which gradually lessened as he got older. At present the veins are seen distended and remarkably distinct all over his limbs; but over the back, chest, and belly the veins assume the look of a network of coarse capillaries. The veins all over the skull are very distinct, and remind one of an anatomical plate, coloured. The fontanelle, which when he was an infant was very large, is not even yet quite closed, and pulsation is still visible in it. The sutures of the head seem normal, and the skull is normally hard. There is not a particle of fat anywhere in the whole of his body.

"The joints are large, but look disproportionate because of the exceeding leanness of his body. The outer condyles of the arm and thigh bones are, however, abnormally large. The movements of the fingers and toes are free enough, but the wrists have a very limited flexion in any direction.

"When an infant up to about two years of age, the head was thinly covered with pale, weak hair. At that time I did all I could to make it grow. I thought I had found parasitic growth on and in the hair, but I suppose now that I must have been mistaken, for there is no sign

¹ 'Archives,' April, 1895.

of hair to be seen on the scalp or on the body, except the very faintest growth of the external ends of the eyebrows. Even lanugo is utterly absent. Not a trace of eyelash.

"His mother has been bald, I understand, ever since she was a girl, and has worn a wig for many years.

"The sisters of this boy are well grown, and have very good hair. One of them had splendid hair,—thick, black, and long. Suddenly her hair began to fall off, leaving large, perfectly bare patches, fortunately cured by Ung. Acid. Carbol., and the hair is excellent again.

"The nails of the feet and hands are very little developed, just visible.

"The teeth are abnormal in number, none are decayed nor have any been lost or extracted. The canine, bicuspids, and incisors are normal, but somewhat irregular, but there are no molars.

"I have written all I can think of. Should there be anything more, I shall be glad to write you.

"I am, yours truly,

"FRED. W. P. JAGO."

When Mr. Hutchinson so courteously permitted me to see this patient he was twelve years older than when this account was written, and had attained a height of 1.096 m., and a weight of 17.25 kilos.

I had been prepared to see a strong likeness between this case and the one which I have described, but I was amazed at the striking personal resemblance of one youth to the other. This doubtless was because the features of the disease and of the person are to some extent identical. Thus in S. B— there was not only the tight scalp and large and prominent veins, the old-looking, lustreless skin, and extremely emaciated face of A. R—; but the same beaked nose, thin lips, protruding eyes, and ill-developed irregular lower jaw. In fact, while neither had the remotest facial resemblance to any of the other members of his respective family (with the one exception I have referred to), and while those families were themselves very unlike,

yet they closely resembled each other. In other respects, too, the likeness was equally remarkable. S. B— came into the room with the same straddling walk, and spoke in the same piping voice as his prototype. His judgment and intelligence were of the same adult character. He was good-natured, quiet, and self-contained in demeanour, sensitive to the opinion of others, and keenly alive to his own personal defects. Like A. R—, he was much affected by extremes of heat and cold. He, too, sometimes sweated profusely from slight causes, and was taken out by his sister in a mail-cart, because he so soon became tired. He was, however, not so short-winded as A. R—, and only on one occasion could he remember being short of breath when not exerting himself, and then he had had to sit up the greater part of the night. He could also take cod-liver oil, though he disliked fat as a rule. His appetite was poor, but he seldom had indigestion. Measles was the only disease with which he was ever laid up.

He also was growing at the rate of about 2·5 cm. a year, his height in June, 1886, being ·907 m., in May, 1894, 1·072 m., and in September, 1895, 1·096 m. His head was 50·8 cm. in circumference, while he measured only 53·4 cm. round the chest on deep inspiration, 45·8 on deep expiration, and 56 cm. round the abdomen at the navel.

The anterior fontanelle was only half closed, and the pulsations of the brain could be easily distinguished, but quite as conspicuous pulsations were also visible on other parts of the vertex. One of these spots was situated on the posterior superior angle of the right parietal bone, close to the lambdoid suture; another was in the corresponding angle of the left parietal, almost in the situation of the posterior fontanelle; and two others were to be seen between this and the anterior fontanelle about 1·8 cm. to the left of the middle line. These four areas were between 2 and 3 mm. in diameter, and the three to the left of the middle line were connected by a curved line of indistinct pulsation, running into the anterior fontanelle.

The pulsations were in a distinct excavation in the bone, and could not be stopped by pressure with the fingers on any one point. The position of the closed lambdoid and coronary sutures could not be detected by touch. There were no bosses of bone, nor wasted areas on the occipital bone.

The scapula was small and the clavicle diminutive. But it was both my own opinion and Dr. Jago's that the head of the radius, though large, was not then disproportionate in size when compared with the other epiphyses, and this was subsequently confirmed on taking a "shadow" photograph of the part. The lower end of the humerus was, however, still relatively large, and the patellæ and both condyles of the femur were conspicuously hypertrophied. The long bones, as a rule, were thin and straight, but each femur had its natural curve exaggerated. Both lower extremities were of the same length, and so too were the upper extremities, which were not so long in proportion as A. R—'s. The backbone also had its natural curve as in A. R—'s case.

On close inspection a feeble growth of colourless hair was found to be present on the backs of the hands and wrists as well as on the head. The trunk and back and sides of the neck were also spotted and pigmented of the same reddish-brown hue, and of the like relative depth, as in the other case. But the colour was deeper, and the face was bronzed and freckled. This last condition I was told was partly due to his having recently gone away for change of air, but was also in part usual with him. The pads of fat near the genitals and at the root of the neck, noticed by Mr. Hutchinson twelve years before, had disappeared.

There was the same inability to fully open the jaw, and the same condition of the tongue as was seen in A. R—. The palate was, however, not abnormal in shape, and the teeth were all of good size and in fair condition, and, though crowded, were by no means so irregular. There were no milk teeth. The alveolar portion of the jaw was

enlarged for their reception, but this not so marked as in my patient.

The meatus of each ear was too swollen to permit of an examination of the drums. With the left ear he heard a watch 25 cm., and with the right 72 cm. away. The right had discharged more or less since he was "quite young."

The palpebral aperture was not narrowed. The right eye showed slight hypermetropic astigmatism in the oblique diameter, and there was hypermetropia of about 2 D. of the left. The optic discs were a little red and their outlines were blurred, but no other abnormal condition was present.

Like A. R—, when he runs he feels a pain beneath the middle of the sternum, but there was no dulness on percussion over the bone. On the contrary, nearly the whole of the chest area was hyper-resonant, and only a little less so over the situation of the heart; the apex beat was best heard 12 mm. outside the nipple line in the fifth space, where there was a soft systolic murmur traceable to the angle of the scapula. A less soft but more distinct systolic bruit could be heard over the aortic area. From this it will be seen that his heart was apparently also in the same condition as in the other case.

In the blood, too, the red discs were too numerous by over 8 per cent., but were not otherwise abnormal. A few minute granular masses could be seen. The white corpuscles were in the proportion of 1 to 236 red. The thyroid gland was easily felt, and was of average size relative to that of the rest of the body. The patellar reflexes were diminished, and no cutaneous reflexes could be obtained. The lymphatic glands could not be felt. The bowels were at one time habitually constipated, but of late they have been relieved regularly. The urine, which was kindly analysed for me by Mr. Hopkins, was found to be normal in every particular.

Remarks.—It should be noticed that in the second of these cases, while some of the symptoms rather suggested

such a cause, there was no direct evidence of persistency of the thymus. In the first it is questionable whether death resulted from thymic or from cardiac asthma. I believe that his habitual dyspnoea was partly the result of pressure of the thymus on the trachea or on the adjacent nerves, for though the thymus was not greatly enlarged relatively to the size of the body¹ it must be remembered that the tiny size of the clavicles greatly narrowed the space between the sternum and the spine. It is my opinion, however, judging from the appearance of the parts after death, that he died of cardiac and not thymic asthma, though a temporary engorgement of the thymus may have acted as a contributory cause. But still more important, probably, was the catarrh and consequent inflation of the stomach, which I attributed to influenza.

His death was singularly like that of many old cardiac cases. It was preceded by a slow general decrease of vigour, by rheumatism, and by the onset of anginal pains, and was precipitated by influenza; and after death there was found extensive atheromatous degeneration of the mitral and aortic orifices with obliteration of the lumina of the coronary arteries. In short, I think there is good evidence in favour of the view that A. R— died of senile decay at the age of seventeen. It is, however, noteworthy that while the heart and blood-vessels were prematurely old, the lungs, if we may judge from the way in which they were affected by disease, were quite the reverse. Thus, they were affected with the infantile disease broncho-pneumonia at the unusually late age of eleven, and it is remarkable that he should have recovered at all from a disease of which the prognosis is so grave in weakly children. But it is probable that the heart mischief had not by that time developed, for the doctor

¹ Triesehtau ('Die Thymusdrüse in norm. u. path. Beziehung') has published a list of cases which show that the hypertrophied gland may be larger than this at the age of eight months, while in the sixth year it may be nearly double the size.

who attended him has no recollection of it. Then again the lobar pneumonia, which he had at the age of sixteen, showed in its rapid and benign course (in spite of the grave complication of atheromatous degeneration of the valves of the heart) that the lungs possessed the recuperative powers of those of a child.

On glancing over the main clinical features of these two cases and of the description of the appearances that were found after death, it is evident that they may all be grouped under two heads. They are indications either of defective growth on the one hand, or of relative increase of growth on the other. The changes which terminate in old age and decay are in abeyance in some parts, while they are accelerated in others.

Evidences of slow or of arrested development were shown in the condition of the bones, especially of the skull, clavicle, and lower jaw, and in the enlargement of the knuckles, but in a lesser degree in nearly all the bones of the body.

There were signs of too rapid development or maturity of the skin, mucous membrane of the tongue and intestines, hair, and nails, and of the brain, liver, and of the blood vascular system. It is noteworthy too that, in testing his sight, dilatation with atropine revealed no concealed visual defect, though he was both hypermetropic and astigmatic.

But it is not only in different tissues or organs that this inequality of nutrition was shown. It was also to be seen in different parts of the same tissue. Thus, while the shafts of the humeri and of the femora were of defective growth, the lower epiphyses were grown quite out of their relative proportion. Indeed, it may be said of the femur that the shaft was the shaft of a young child while the condyles were those of a youth. This feature is also well shown in the case of the teeth in A. R.—. In the same jaws were unshed milk teeth crowded alongside average-sized or unusually large permanent teeth. The tongue, too, was more developed on one side than on

the other, and one half of the skull was larger than the other half. In one ear of A. R— it was interesting to see an incus of adult size taking up so much room in the ear of a child. In the hypertrophied thymus, again, we have an instance of arrested development of an infantile structure and its hypertrophy in the same part.

These are, so far as I am aware, the only two cases of this affection which have hitherto been described. But the condition is so singular, the life-history of one case so complete, and that of the other so evidently of the same species, that there can, I think, be little hesitation in regarding it either as a distinct disease, or such a striking variation from some known disease as almost to merit a name of its own. I will venture, for the purposes of this paper, to give it the provisional name of micromegaly. I am fully aware of the objection to a word which is made up of two adjectives, but the fact that those two adjectives express the opposite qualities of smallness and largeness is, in my opinion, a point greatly in its favour instead of the reverse, for the disease manifests itself in these two directions. It connotes in one word the two great features of the collection of changes and symptoms which together constitute the disease. Above all it serves to indicate its connection with a disease to which it is apparently closely allied. I allude to acromegaly. At first it may appear that there can hardly be two diseases which are more widely different. Indeed, in some respects the features of the one disease are the exact contrary to those of the other. Thus the patient with acromegaly has thick lips, a large tongue, and heavy jaws, while these micromegalics have thin lips, small tongues, and ill-developed jaws. In acromegaly the collar-bone is often hypertrophied, while here its development is arrested. In acromegaly the hair is abundant, and the skin is thick (pachydermatous), and becomes loose, yellow, or brown, and scaly; in these cases of micromegaly both the skin and its appendages are atrophied almost from the beginning. In acromegaly there is both atrophy and hypertrophy of the sexual

organs ; in these cases they are apparently unaffected. In acromegaly the intelligence may become childish while the body takes on the proportions of a giant ; in these instances the reverse is the case, the mind may be precocious while the body is stunted. But these differences are not indications of a want of connection between the two diseases. On the contrary, they show their relationship—that the one is in some way complementary to the other.

In the above comparison the widely different effect of each disease on the same parts has been shown. In other respects, though the same parts are not affected, the method of the disease may also be shown to be the same, *e. g.* while in acromegaly it is the lower ends of the radii and tibiae which are enlarged, in these cases it is the lower ends of the humeri and femora. Here, too, an associated cause is apparently at work in the two diseases, though the effects are different. Both are trophic diseases. In both there is relative over-development, on the one hand, going on side by side with arrested development on the other.

Another factor of importance to be taken into consideration in estimating the relations between the two diseases is the variation that occurs in the symptoms of acromegaly in different cases. The same parts are not always affected, and atrophy in one case may be hypertrophy in another. For example, the muscles are wasted, as a rule, and correspondingly feeble, but sometimes they are quite Herculean in their proportions and strength. For this reason, also, too much importance ought not to be attached to the hypertrophy of the pituitary body in the one disease and not in the other, for it has been shown that in more than 20 per cent. of the autopsies that have been made on acromegalics, no such condition was present. It is not an essential part of the disease.

It can also be shown that a few of the symptoms of the two diseases are alike, and some of these are of importance in their bearing on their mutual relations.

One of the first names that was given to acromegaly was "exophthalmic cachexia" in recognition of the prominence of the eyeballs that is one of the features of the disease, and of the debility, which is another. Both of these symptoms were also present in each of the two cases of micromegaly that have just been reported. Much of the weakness of which acromegalics complain is due to the emaciation of the muscles; and the same feeble muscular power, with wasting, is also present as a symptom of micromegaly. The marked pigmentation of the skin so often met with in the former disease reminds one of that pigmentation which was present in both cases of the latter disease. One of the features of these two cases of micromegaly is the large size of the cartilages of the nose. This corresponds with the hypertrophy of the same tissue in acromegaly. The alteration in the tone of the voice, the atrophy of the mammary glands and nipples, the increase of perspiration, the flattening and atrophy of the nails, the diminution or absence of patellar and skin reflexes, are also common to both diseases. A condition of the blood-vessels is sometimes present in acromegaly, which is suggestive of early atheroma, while the heart is enlarged. In other cases the thymus has been found hypertrophied. Both these conditions are present in these instances of micromegaly. In short, in acromegaly as in micromegaly, the clinical features of the disease are hypertrophy and atrophy, irregular development, and premature decay.

Is it possible that micromegaly is acromegaly modified by age? Do they bear the same relation that congenital syphilis does to acquired syphilis; or are they opposed as cretinism is opposed to exophthalmic goitre? *A priori* it seems reasonable that a trophic disease will show very different manifestations when it appears respectively in infancy, at puberty, or when the body is fully developed. In an interesting article by Dr. Woods Hutchinson giving the account of the autopsy of a giantess of about eighteen, who was 6 feet 7½ inches high, some of the features of the disease were wasting of the mammae,

sparseness of hair, extreme emaciation, and debility ending in death. A reproduction of a photograph of this giantess is printed with the article. In it she is represented as standing beside her two sisters, one of whom appears to be a full-grown woman, while the other, to judge by her size, is a child of less than twelve years old. Nothing is said of this girl in the paper, but elsewhere I have found that she was in reality the eldest of the three, and that she was thirty-two years of age when the photograph was taken. Now there is nothing in this dwarf to remind one of the above cases, with the important exception of her hands, which show an enlargement of the knuckles similar to that which obtained in both Mr. Hutchinson's case and my own. But surely it cannot be due to mere chance that two such rare conditions as true gigantism and true dwarfism should occur in the same family. The gigantism in this case was due to acromegaly. Acromegaly shows hereditary tendencies. Is it not probable that the dwarf also owes her condition to the same disease? ¹

Does acromegaly of infancy tend to produce dwarfism, and of puberty gigantism?

Are all dwarfs of the "Tom Thumb" type micromegalics? A few years ago I saw the Mongolian giant Chang when he was being shown in company with a dwarf. Is it possible that both the giant Chang and the feeble little midget who stood on his hand were instances of the same disease?

As a final speculation it may be asked, is micromegaly in its slight or less marked forms, more prevalent than is supposed. Mr. Hutchinson has done much to call attention to the great utility of the study of rare diseases in view of their commoner occurrence in slight or atypical forms, and of the light they throw on other diseases. I call to remembrance a case of a child of eight years of age who died from the results of middle ear disease while under my care. Though well cared for, she was thin, weakly,

¹ I have not yet been able to obtain confirmation of this relationship.

and puny, and of very feeble muscular development, but mentally she was unusually sharp for her age. Was this an instance of micromegaly of aberrant type, and if so, are there many such cases of mild or partial forms of the disease that have hitherto been unrecognised?

(For Report of the Discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 4.)

DESCRIPTION OF PLATE V.

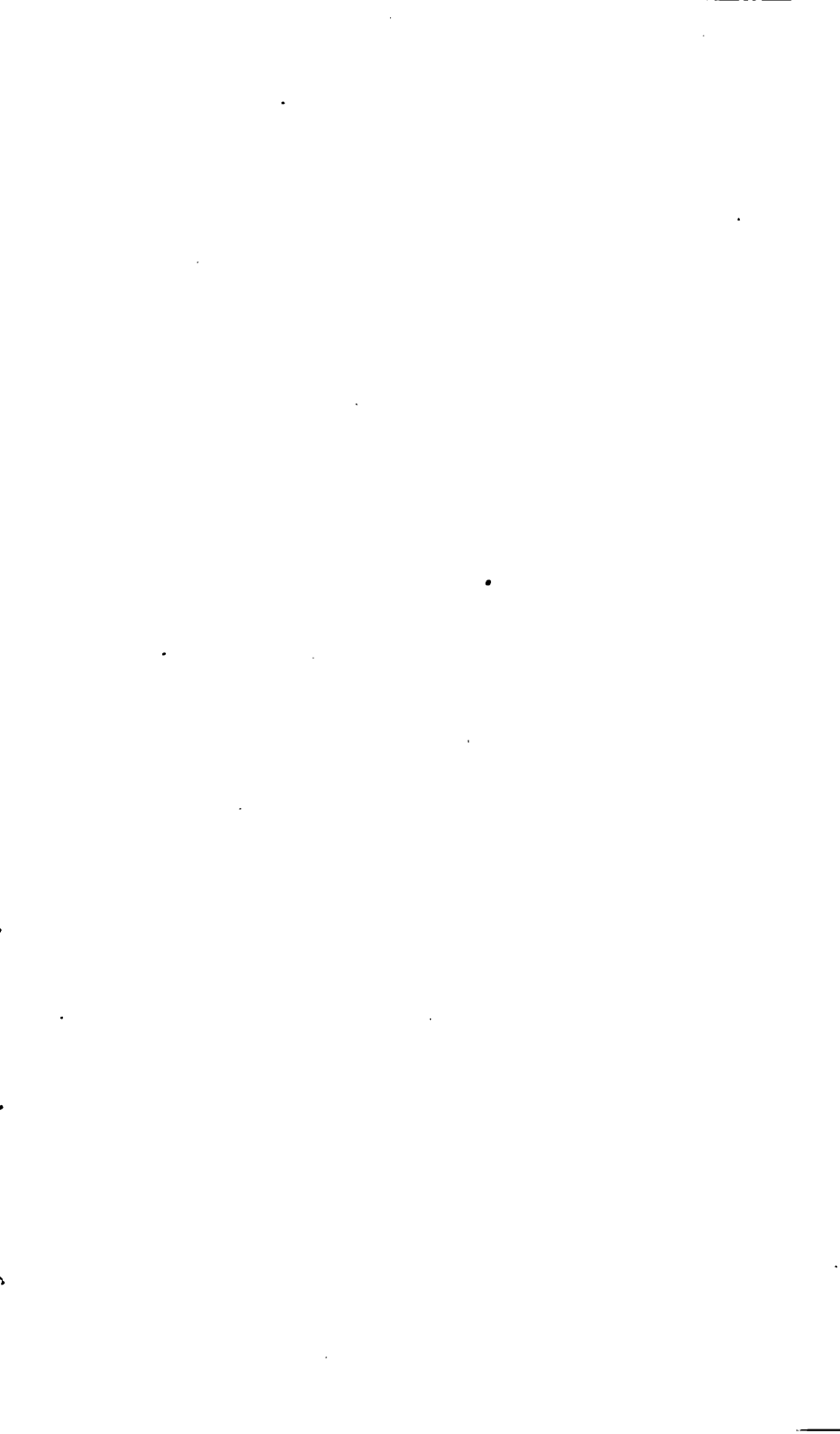
**On a Condition of Mixed Premature and Immature Development
(HASTINGS GILFORD).**

A. R—, at different stages of disease, showing rapid and progressive general maturity.

Age one and a half years.







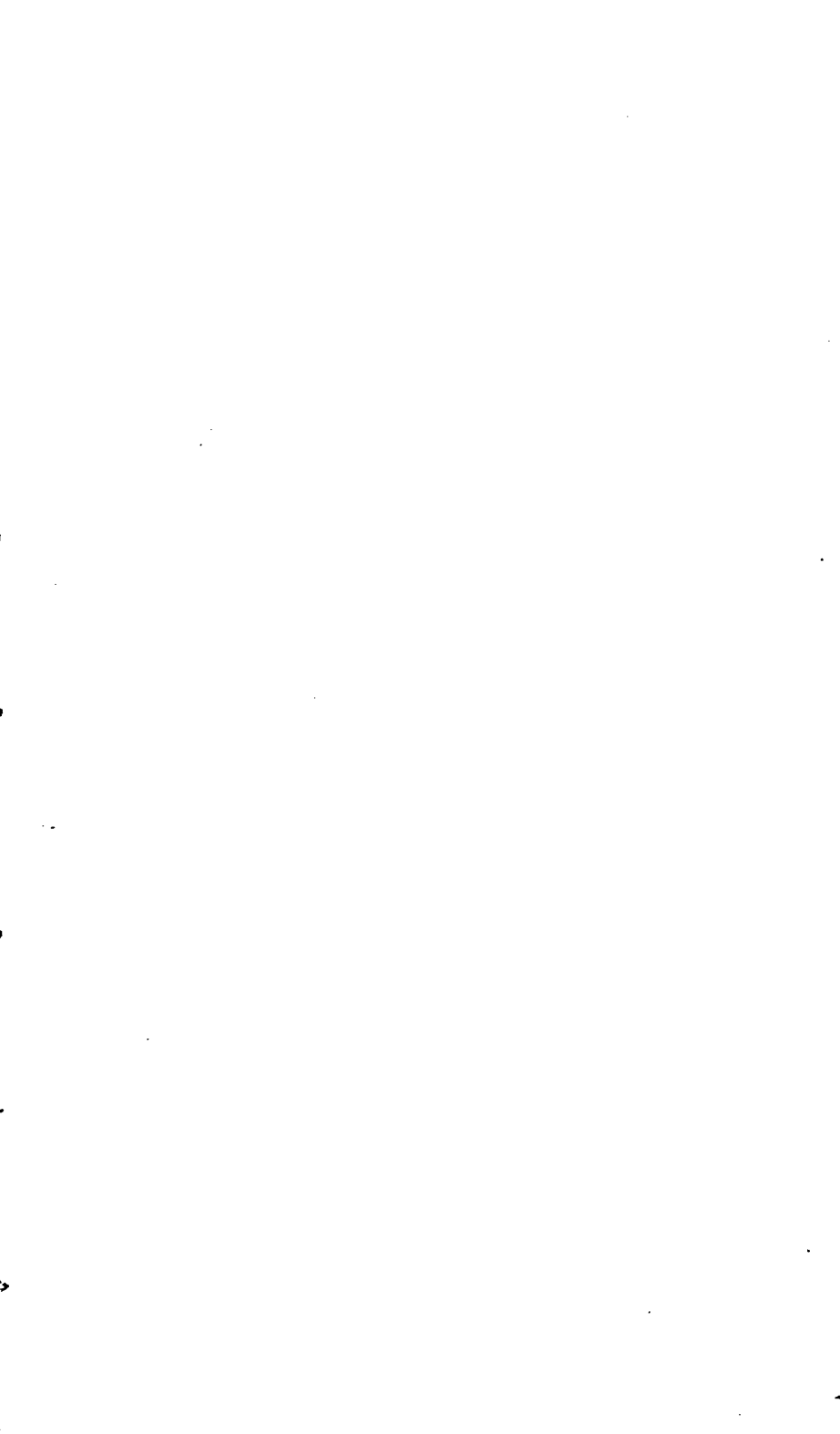
DESCRIPTION OF PLATE VI.

**On a Condition of Mixed Premature and Immature Development
(HASTINGS GILFORD).**

Age seven years.







DESCRIPTION OF PLATE VII.

**On a Condition of Mixed Premature and Immature Development
(HASTINGS GILFORD).**

Age twelve years.







DESCRIPTION OF PLATE VIII.

**On a Condition of Mixed Premature and Immature Development
(HASTINGS GILFORD).**

Age fifteen years.





DESCRIPTION OF PLATE IX.

**On a Condition of Mixed Premature and Immature Development
(HASTINGS GILFORD).**

**Age seventeen years. Normal hand introduced to show
relative size.**



DESCRIPTION OF PLATE XI.

**On a Condition of Mixed Premature and Immature Development
(HASTINGS GILFORD).**

**Mr. Hutchinson's case, S. B—. Age fifteen and a half years.
Normal hand introduced to show relative size.**

Front view.





ON A
FORM OF CHRONIC JOINT DISEASE
IN CHILDREN

BY

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THE occasional occurrence in children of a disease closely resembling the rheumatoid arthritis of adults has been recognised for several years. The identity of the disease seen in children with that in adults has never, so far as I am aware, been called in question.

The purpose of the present paper is to show that although the disease known as rheumatoid arthritis in adults does undoubtedly occur in children, the disease which has most commonly been called rheumatoid arthritis in children differs both in its clinical aspect and in its morbid anatomy from the rheumatoid arthritis of adults; it presents, in fact, such marked differences as to suggest that it has a distinct pathology.

The cases hitherto grouped together as rheumatoid

arthritis in children include, therefore, more than one disease; and it will be shown that there are at least three distinct joint affections which have thus been included under the one head, Rheumatoid arthritis.

The paper is based on a study of twenty-two cases, almost all of which have been in the Hospital for Sick Children, Great Ormond Street. Nineteen of these I have had under personal observation.

It will be necessary first to describe briefly the disease to which I have referred as the subject of this paper, and subsequently to point out the features of its clinical course and morbid anatomy, wherein it differs from the rheumatoid arthritis of adults.

The disease may be defined as a chronic progressive enlargement of joints, associated with general enlargement of glands and enlargement of spleen.

The onset is almost always before the second dentition; ten out of twelve cases began before the age of six years, and of these eight began within the first three years of life: the earliest was at fifteen months.

Girls are more commonly affected than boys; seven of the twelve cases were girls, five were boys.

The onset is usually insidious; the child, if old enough, complains of stiffness in one or more joints, which slowly become enlarged, and subsequently other joints become affected; but occasionally the onset is acute, with pyrexia and, it may be, with rigors.

I wish to lay some stress on the character of the enlargement of the joints. It feels and looks more like general thickening of the tissues round the joint than a bony enlargement, and is correspondingly smooth and fusiform, with none of the bony irregularity of the rheumatoid arthritis of adults.

The absence of osteophytic growth and of anything like bony lipping, even after years have elapsed since the onset, is striking.

There is, I believe, never any bony grating, although creaking, probably either of tendon or of cartilage, is

frequently present. There is no redness or tenderness of the joints, except in very acute cases. The absence of pain is generally striking, but it may be present in slight degree, especially on movement. Limitation of movement, chiefly of extension, is almost always present; the child may be completely bedridden owing to more or less rigid flexion of joints.

The extensive deformities of the hands described by Charcot as occurring in the rheumatoid arthritis of adults (*'Maladies des Vieillards,'* 2nd edit., p. 201) are unknown to me in this disease. Those most common so far as I have seen are, flexion of the wrist with slight deviation of the hand to the ulnar side, and slight flexion of the proximal, combined sometimes with slight flexion of the distal, inter-phalangeal joint. Rarely there is slight hyperextension of the metacarpo-phalangeal or proximal inter-phalangeal joint. The fingers may deviate very slightly to the radial side, but more often the deviation is at the proximal inter-phalangeal joint, and may be to either side; indeed, both directions may be seen in one hand. Adduction of the thumb was marked in one case.

The joints earliest affected were usually the knees, wrists, and those of the cervical spine; the subsequent order of affection being ankles, elbows, and fingers. The sterno-clavicular joint was affected in two out of twelve cases; the temporo-maxillary in three. The affection is symmetrical. There is no tendency to suppuration nor to bony ankylosis. The muscles which move the diseased joints show early and marked wasting, which contrasts often strongly with the good nutrition of the rest of the body.

The electrical reactions both to faradism and galvanism were brisk in three cases tested, but not otherwise altered.

Perhaps the most distinctive feature in these cases is the affection of the lymphatic glands. The enlargement is general, but affects primarily and chiefly those related to the joints affected. The glands are separate, rather hard than soft, not tender, and show no tendency to break down. They may become so large as to be visible, but

more often do not become larger than a hazel-nut. The enlargement seems to bear a definite relation to the progress of the disease in the joints. Slight affection of the glands is found very soon after the first symptoms of the joint affection, and as the latter increases the glands become larger. If the joint affection subsides, the glands become smaller, increasing again in size if the joints become worse.

The glands most affected are the supra-trochlear, those along the brachial artery, and those in the axilla, also those in Scarpa's triangle, and deep in the iliac fossa along the iliac artery, and those in the posterior triangle of the neck. In one case I thought that the popliteal glands were enlarged, and in two cases there was some evidence clinically of enlarged mediastinal glands. I have never been able to make out enlargement of mesenteric glands, but in one of the autopsies which I shall mention, the glands in the hilum of the liver were found enlarged.

It will thus be seen that the enlargement is general; and I may add that it is constant; it was found in all the twelve cases mentioned.

Enlargement of the spleen is also a striking feature of these cases. It is, of course, not always easy to be certain of splenic enlargement, but it was definite and considerable in nine out of the twelve cases, the edge of the spleen being felt one to two fingers' breadth below the costal margin. The enlargement of the spleen seems to be roughly proportionate to that of the glands, and like that of the glands has been observed to increase as the joint condition became worse.

The heart shows no evidence of valvular disease, but hæmic bruits were detected in some of the cases.

There were some physical signs suggestive of adherent pericardium in two of the twelve cases, and in three other cases adherent pericardium was found quite unexpectedly at the autopsy.

Anæmia is generally present to some extent, but is seldom profound; the face has often a curious waxy pallor with

flushed cheeks. The blood shows only moderate diminution of red corpuscles in most cases; in some, however, there is disproportionate deficiency of hæmoglobin.

A curious symptom noticed in four cases out of the twelve, was slight prominence of the eyes, hardly enough to call exophthalmos, but enough to be noticeable. The thyroid seemed normal in the cases which I examined.

Sweating is often profuse, and not related to temperature. The temperature seems to be of two varieties: the one shows periods, generally lasting only a few days, of pyrexia followed by a longer interval of apyrexia; the other shows more or less continuous slight pyrexia. The pyrexial attacks occasionally show a curious regularity in their recurrence. One or two cases showed sudden attacks of hyperpyrexia, lasting one hour or two, and then subsiding rapidly. The pyrexial periods are not usually associated with any clinically demonstrable exacerbation of the joint trouble, nor indeed is it possible usually to find any definite cause for the fever.

I have made a detailed study of the urine in these cases, and find that the urea, uric acid, phosphoric acid, and chlorides each show considerable variations, but that these variations are within the limits of health, as shown by a series of analyses of the urine from healthy children. The urine in other respects also was normal.

A remarkable feature in these cases is the general arrest of development that occurs when the disease begins before the second dentition. A child of twelve and a half years would easily have been mistaken for six or seven years, while another of four years looked more like two and a half or three years.

The arrest is, however, of bodily rather than of mental development, and hence although backward in some respects from the enforced absence from school, the child often appears by comparison with its size to be rather precocious than backward.

The course of these cases is slow. Improvement may occur for a time under treatment or spontaneously, but the

disease soon progresses again until a condition of general joint disease is reached which seems to be permanently stationary. The disease is not in itself fatal; the few deaths that have been recorded were due to complications.

Curiously enough, some accidental complications have been followed by marked improvement; thus I have known measles, scarlet fever, and catarrhal jaundice, to be each followed by distinct improvement of the joint symptoms.

The ætiology of the disease is very uncertain. A careful investigation of the family history in ten cases gave the following figures: phthisis in five, acute rheumatism in four, rheumatoid arthritis in one (a grandmother), gout in none, syphilis in none. The frequency of acute rheumatism and phthisis here cannot be considered to have any special significance; a similar investigation of fifty consecutive non-rheumatic, and also of fifty non-tubercular cases in the Hospital for Sick Children gave respectively 44 per cent. and 46 per cent. as the number of times that acute rheumatism and phthisis occurred in the family history.

No relation to diet in infancy could be shown. Poverty and exposure to insanitary conditions seemed to play little if any part in its causation.

The morbid anatomy of this disease is gathered from three post-mortems. (I unfortunately did not see these myself, so that my information is obtained from the post-mortem records, corroborated by those who saw the autopsies.) For permission to make use of the record of one of these cases I am indebted to the kindness of your President, Dr. Dickinson.

The joints show marked thickening of the capsule and of the connective tissue just outside this. There is also thickening and vascularisation of the synovial membrane, and fibrous adhesions are sometimes present.

The cartilage may be perfectly normal, as in two cases that had lasted nearly one and a half years; but in a case that had lasted three years it showed pitting of its surface as if from pressure, with little processes of the thickened synovial membrane fitting accurately into the pits, which

were situated chiefly at the margin of the cartilage ; otherwise, however, the cartilage was healthy,—there was no fibrillation, no osteophytic change, no exposure or eburnation of bone.

The enlarged glands appear normal on section, or show small ecchymoses in their substance.

The spleen weighed in each case about 5 ounces, so that it was considerably enlarged ; it was firm, and appeared normal on section.

In each case the pericardium was universally adherent ; there were also pleural adhesions. There was no endocarditis certainly in two cases, but in the third the mitral valve was perhaps a little thickened.

The following case, under the care of Dr. Lees at the Hospital for Sick Children, Great Ormond Street, may serve to illustrate some of the points mentioned.

Alice C—, at the age of two years and four months began to limp in walking, and it was noticed that the ankles were swollen. A week or two later the elbows, and then the knees, became stiff and swollen ; stiffness of the neck also was noticed almost from the first onset. The joints affected became steadily worse, and the child quickly lost the power of standing.

There had been no previous illness, except whooping-cough ; the child had been carefully hand-fed after it was four weeks old ; up to this time it had been breast-fed. There had been no privations, but the house was damp. The family history showed nothing of importance except a doubtful history of rheumatism in two maternal uncles.

When first seen at the age of three and a half years, the child was fairly nourished, with round face and slightly prominent eyes. All the joints of the limbs were affected ; extension of the knees, hips, and elbows was considerably limited, there was thought to be a little fluid in the knees, ankles and wrists, and there was obvious fusiform elastic thickening of all these joints except the shoulders and hips. I could not find any definite bony enlargement, and there was no grating. The sterno-clavicular and temporo-

maxillary joints, and dorsal and lumbar spine, were unaffected.

Gland enlargement was marked; the axillary, supracondylar, cervical, iliac, and inguinal were affected; the axillary were easily visible. The glands were hard, separate, and not tender.

The spleen was one finger's breadth below the costal margin. Heart and lungs were normal.

For nearly two years the child has been in the Hospital for Sick Children, and the joint condition has slowly become worse (as will be seen from the photograph shown), so that the child is unable even to turn herself in bed. The progress of the disease has, however, not been steady; at one time there was a slight improvement in the joint condition, and synchronously with this the glands and spleen became much smaller.

The temperature chart has shown the recurrent attacks of pyrexia described above.

Having sketched the clinical aspect and morbid anatomy of the disease, I wish now to draw attention to the points in which the condition differs from the rheumatoid arthritis of adults.

The clinical features of the joint affection cannot be considered distinctive, but it may be pointed out that the fusiform enlargement which feels like extra-articular thickening of soft tissues, and the absence of bony lipping and grating, even after the disease has lasted some years, are very unlike the irregular bony enlargement of joints found in the advanced rheumatoid arthritis of adults.

Pathologically, however, the joints show marked differences. In the children's disease there is complete absence, even in an advanced case, of the cartilage changes which are seen quite early in the rheumatoid arthritis of adults. In the children's disease, also, there is a very considerable thickening of the capsule, and of the connective tissue just outside this, which is a much less prominent feature in the disease of adults.

On the enlargement of glands I lay great stress. It is,

I think, one of the most important points of distinction clinically between this disease and rheumatoid arthritis. It is, as far as I know, never found in the rheumatoid arthritis of adults, whereas it is a constant symptom in the disease of children here described.

The enlargement of the spleen, associated with the glandular enlargement, is another important distinction, and like the preceding symptom is, I believe, unknown in adults.

Other minor differences are the following :

The incidence on the sexes is different. The proportion of females to males affected by rheumatoid arthritis in adult life was found by Sir A. Garrod to be 5 : 1, whereas in the disease described above the proportion is barely 1·5 : 1 ; my numbers, however, are so small that no great weight can be attached to this difference.

The order of affection of joints is different. In adults rheumatoid arthritis affects the small joints of the hands quite early, and often begins here ; whereas the disease of children begins nearly always in the knees or wrists, and the fingers remain often free for months or even years. The very early and almost constant occurrence of affection of the cervical spine is also, I fancy, far more common in the disease here described.

Lastly, the occurrence of adherent pericardium, certainly in three, probably in five cases out of twelve, with no clinical evidence of endocarditis in any case, and only a slight thickening of the mitral valve of very doubtful significance found post mortem in one case, and also the occurrence of pleurisy in four out of twelve cases, suggests some peculiar liability of children with this disease to inflammation of serous membranes, a liability which is not shared by the rheumatoid arthritis of adults.

From the foregoing it is seen that there are well-marked clinical and pathological differences between the rheumatoid arthritis of adults and the disease of children above described. But when it is suggested that the two conditions are therefore different diseases, it may be objected

that the points of distinction mentioned may all be due merely to the difference of age.

This objection seems to me sufficiently answered by the fact that there occurs in children, rarely indeed, but only rather more rarely than the disease here described, a condition which appears to be identical in every respect with the rheumatoid arthritis of adults.

I shall now deal briefly with the second point, namely, that the cases hitherto grouped together as rheumatoid arthritis in children include at least three distinct conditions.

The least rare is the disease already described, which for the reasons I have given should not be called rheumatoid arthritis.

Next in frequency is a joint affection which cannot be distinguished from the rheumatoid arthritis of adults. It is unnecessary to describe it. It presents the same general enlargement of joints, with subsequent bony thickening and lipping, and bony grating as in adults. There is no enlargement of lymphatic glands or spleen, and no evidence of pericarditis.

Of the twenty-two cases, six were of this nature, and the age at onset was generally rather higher than in the preceding disease; two began before the second dentition, one at one year and eight months.

(In the photograph shown by kind permission of Dr. Cheadle, the contrast is seen between the fusiform enlargement of the phalangeal joints in the first disease, and the bony enlargement of rheumatoid arthritis.)

The third condition that has, I venture to think, been confused with rheumatoid arthritis is a form of rheumatism. It is extremely rare. I have seen only one case. It was in a boy, aged five years, under the care of Dr. Lees at the Hospital for Sick Children.

At the age of three and a half years the fingers became stiff, apparently from the formation of fibrous nodules over the tendons. Then the elbows and wrists slowly enlarged, and their movements became limited. There was also some

stiffness of the neck. There was no tenderness of joints, and no acute illness at any time. Seen one and a half years later, the elbows and wrists showed firm thickening, very suggestive of extra-articular fibrous thickening. There was none of the fusiform elastic enlargement of the phalangeal joints which was seen in all the advanced cases of the disease described in the earlier part of this paper; indeed, there was no evident affection of the phalangeal joints, although there was marked limitation of movement, the terminal phalanges being flexed, and some of the proximal phalanges hyper-extended. There was a loud apical systolic bruit; there was no enlargement of spleen or glands. Subcutaneous fibrous nodules were present on the fingers, elbows, and head.

In this particular case, although there was no history of acute rheumatism, the association of a cardiac bruit with fibrous nodules was, I think, sufficient proof of the rheumatic nature of the joint affection; but in cases where such additional symptoms were not present to aid the diagnosis, the disease might easily be mistaken for an early rheumatoid arthritis. There can, I think, be no doubt that it is identical with the disease described by Jaccoud (art. "Polyarthrite Déformante," 'Pathologie Interne,' 1871) as chronic fibrous rheumatism.

It is particularly interesting to note that although Jaccoud described the condition as a disease of young adults, and occurring after repeated attacks of rheumatic fever, in the case I have mentioned the disease began at three and a half years of age, and there was never any acute rheumatism.

The points of difference between this and the preceding conditions are the following:—The evidence of rheumatism, and this is the most diagnostic feature when present, distinguishes it from both the previously described diseases. The absence of bony thickening and of bony grating, and the character of the joint enlargement, which strongly suggests extra-articular fibrous thickening, and I think also the much less likelihood of affection of the small joints

of fingers and toes, distinguish this disease from rheumatoid arthritis in children; while the absence of gland and spleen enlargement, and probably here also the less likelihood of affection of the small joints, distinguish it from the disease described in the first part of this paper.

In conclusion, I should like just to mention another affection which I have known raise the question of rheumatoid arthritis in a child. It is a rare form of syphilitic joint disease. It shows, in addition to the commoner chronic effusion with thickening of capsule in medium-sized and larger joints, definite bony thickening and lipping, which affects also the smaller joints; this osteophytic change may even simulate Heberden's nodes, as I have seen in a boy six years old, in whom some of the distal phalanges showed lateral thickenings very like Heberden's nodes. They were, however, less regular in their distribution, for they occurred not only on the distal, but also on one or two of the proximal phalangeal joints, and the toes showed similar nodosities. In this boy the larger joints showed chronic effusion, with some thickening of surrounding soft tissues, and there was a gumma over one ulna, and further evidence of syphilis was found in old iritis. There was no enlargement of glands or spleen.

It may be useful to sum up the conclusions arrived at in this paper.

There is a disease, occurring in children, and beginning before the second dentition, which is characterised clinically by elastic fusiform enlargement of joints without bony change, and also by enlargement of glands and spleen.

This disease has hitherto been called rheumatoid arthritis, but it differs from that disease in adults, clinically in the absence of bony change, even when the disease is advanced, and in the enlargement of glands and spleen, and pathologically in the absence, even in an advanced case, of the cartilage changes which are found quite early in that disease, and also in the absence of osteophytic change.

These differences are not to be attributed merely to modification of disease by difference of age, as there occurs

also in children a disease in every respect identical with the rheumatoid arthritis of adults.

Under the head of Rheumatoid Arthritis in children, at least three conditions have been confused which are both clinically and pathologically distinct, namely:—(1) the joint disease described in the present paper; (2) a disease identical with the rheumatoid arthritis of adults; (3) a disease probably identical with that described by Jaccoud as chronic fibrous rheumatism.

Before ending this paper, I wish to express my gratitude to Dr. Barlow, Dr. Lees, Dr. Penrose, and Dr. A. E. Garrod, not only for kind permission to use their cases, but also for allowing me to bring forward this subject. They have, I know, taken great interest in these cases for several years, especially in the disease mentioned in the earlier part of this paper, and therefore several of the points which I have described had been already observed by them. I have to thank them for drawing my attention to some of these points, and for allowing me to make use of their observations. To Dr. Garrod I am indebted also for many valuable criticisms and suggestions.

Since the above paper was written, two French observers, Chauffard and Ramond ('*Revue de Méd.*,' May, 1896), have described occasional enlargement of the lymphatic glands in adults with an acute form of rheumatoid arthritis, which they distinguish as "infective arthritis;" similar cases have been observed by Drs. Bannatyne and Wohlmann of Bath ('*Lancet*,' April, 1896). The disease, however, which I have described seems to differ in several points from the conditions described by these observers.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 11.)

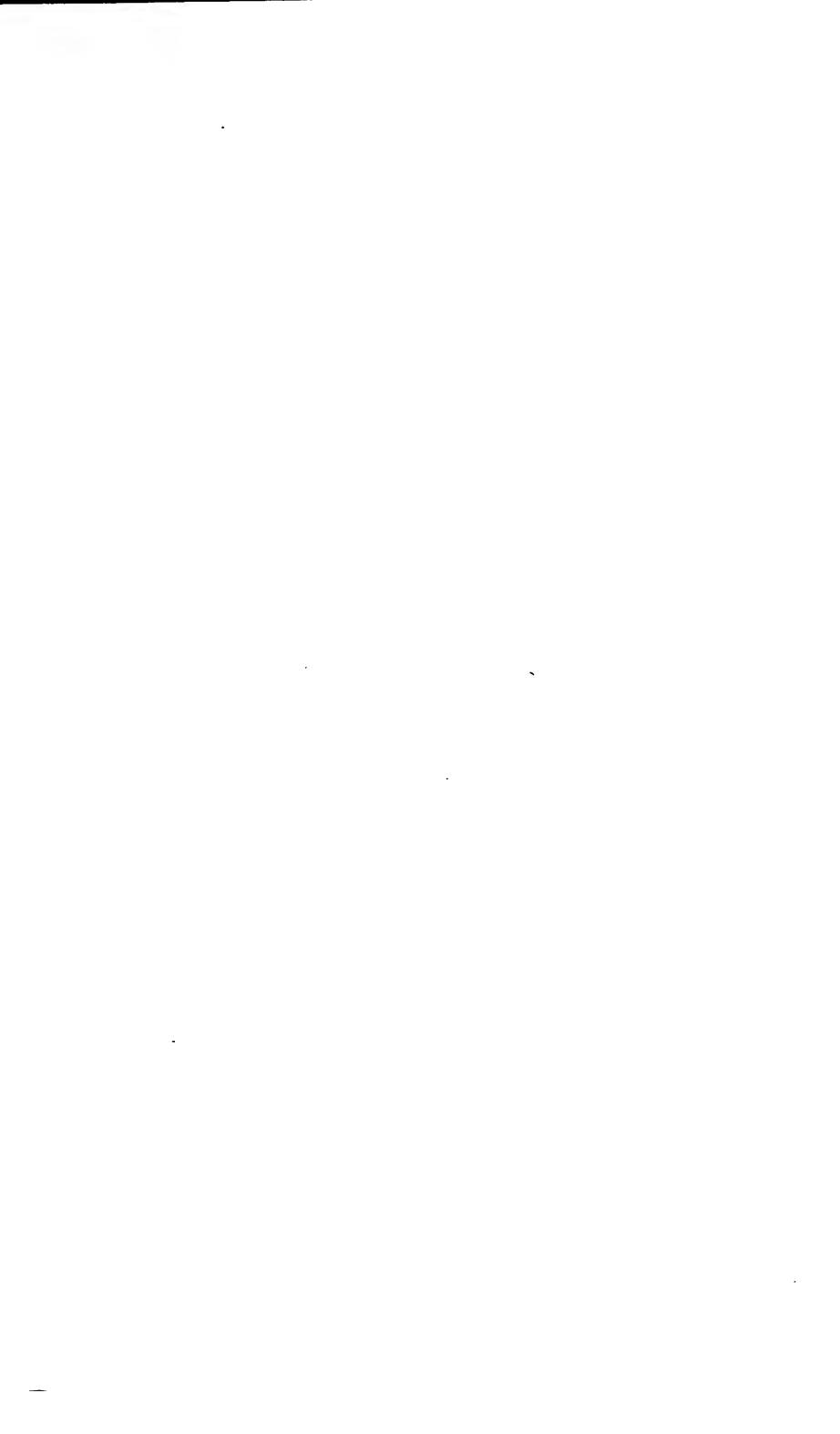
DESCRIPTION OF PLATE XII.

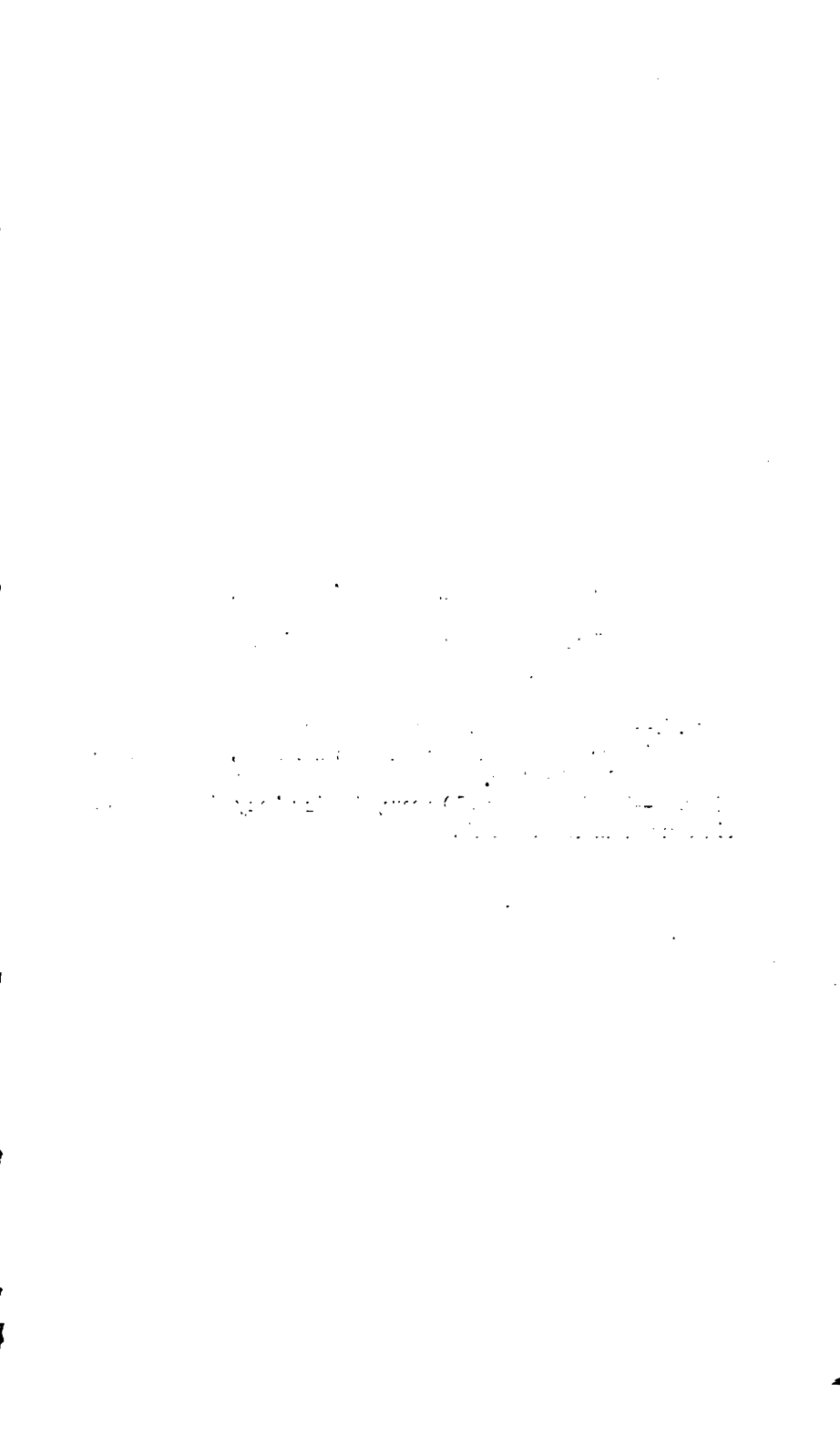
On a form of Chronic Joint Disease in Children
(Dr. GEORGE F. STILL).

Chronic arthritis, with enlargement of glands and spleen.

Alice C—, æt. 4 years; showing tendency to fixation of joints in position of flexion.







DESCRIPTION OF PLATE XIII.

On a form of Chronic Joint Disease in Children
(Dr. GEORGE F. STILL).

FIG. 1.—Hands of girl *æ*t. 7 years, showing fusiform enlargement of joints in the form of chronic arthritis described, with enlargement of glands and spleen.

FIG. 2.—Hand of boy *æ*t. 10 years, showing bony thickening of joints in rheumatoid arthritis.



Fig. 1.

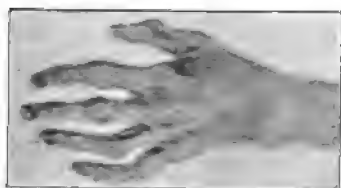


Fig. 2.



DESCRIPTION OF PLATE XIV.

**On a form of Chronic Joint Disease in Children
(Dr. GEORGE F. STILL).**

Chronic arthritis, with enlargement of glands and spleen.

FIG. 1.—Girl *æt.* 3½ years; disease began at 2½ years.

FIG. 2.—Jane R—, *æt.* 4 years; disease began at the age of 15 months.



Fig. 2.



Fig. 1.



ON A CASE
OF
AMNESIA AND OTHER SPEECH
DEFECTS
OF EIGHTEEN YEARS' DURATION
WITH AUTOPSY

BY
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Received October 1st—Read November 24th, 1896.

I HAVE ventured to bring this case before the Society as it is one which possesses considerable interest from a clinical point of view, and has important physiological and psychological bearings. It is remarkable also for the constancy of the speech defects over a long series of years, as well as on account of the difficulties in interpretation, looking to the pathological conditions revealed by the autopsy.

It seems almost certain that the lesion found was far more extensive than that which could have existed in the early days of the patient's illness—in other words, that

the lesion must have been progressive, although the patient's symptoms were of a comparatively stationary type. Two consequences follow from this: there is, first, the impossibility, unfortunately, of accurately localising and defining the extent of the lesion that gave rise to a most interesting type of speech defect; and, secondly, there is the difficulty in understanding how the complete destruction of certain convolutions, such as the hinder two thirds of the upper temporal, and the supra-marginal and angular gyri, could have occurred, as they did, on the left side of the brain of a right-handed man, without producing their usual results in the form of "word-deafness" and "word-blindness."

The total symptoms presented by the patient are divisible into two categories, namely, (1) those more immediately pertaining to the right-sided hemiplegia; and (2) those relating to the mental condition with associated speech defects. In the former group there was some evidence of progression; in the latter there was none, and scarcely any evidence of variation even, over a long series of years. This latter will be seen to be a fact of extreme importance in view of what was found at the autopsy. It will be necessary, therefore, to substantiate it by ample evidence, which is fortunately possible seeing that the patient was not only examined by myself, and his case demonstrated to students at very frequent intervals, but was also submitted to careful examination by several of my house physicians, as will be shown by extracts from my case-books of different periods.¹ On account of the interesting nature of his case the man was purposely kept under observation, and encouraged to come to the Hospital from time to time. He was first seen three months after the onset of an apoplectic attack which had left him partially paralysed on the right side.

¹ The house physicians, from whose notes I shall have to quote, were: Dr. Boyd Joll (1878), Dr. Beevor (1879), Dr. Halliburton (1882), Dr. Sidney Martin (1883), Dr. Rüffer (1886), Dr. R. H. Castellote (1894-5), Dr. Harold Way (1895-6).

Thomas Andrews, æt. 32, a tin-plate worker, had always been temperate, and had never suffered from syphilis. He had a slight fright about the middle of December, 1877, and two days after, as he was going out to work, about 8 a.m., he suddenly fell backwards on a chair. He was not convulsed, and did not lose consciousness, but he lost power in the limbs of the right side and "did not speak coherently after the commencement of the fit." The limbs seem to have been completely paralysed at first, and there was incontinence of urine and fæces for four or five days. "He also had headache which persisted for some time."

He was admitted under my care into University College Hospital on March 12th, 1878, by which time he had recovered a good deal of power in the paralysed limbs. There was no cardiac bruit at the base or apex. The right angle of the mouth was somewhat lower, and not raised so well as the left. The tongue was protruded considerably to the right. The grasp of the right hand was extremely feeble. He could raise the right foot two feet off the bed, and could resist flexion of the right knee with considerable force. The movements at the right ankle-joint were greatly impaired, though not altogether absent. He walked with difficulty, dragging the right foot somewhat. Tactile impressions were badly appreciated, and painful impressions still more so, on the limbs of the right side. (No mention is made of trunk or face, in this respect.) Sight and hearing good.

He continued to improve slowly, and his condition as regards speech and related powers were thus described on April 2nd:—"He recognises common objects, but cannot name them; repudiates a false name, and recognises the real one at once when he hears it. Can never remember his own name till it is suggested to him. On being asked to repeat it, after a few trials which vary each time, he pronounces it 'Anstruthers' or 'Anstrews.' His first name seems to come more readily, and he can often attempt this without prompting. But

either after it has been repeated to him, or when he says it spontaneously, he pronounces it 'Touvers.' The letter *l* is difficult for him to utter; sometimes he pronounces it like a *d*, and at others like a *v*. He has been taught to count, and can fairly pronounce the numerals from one to twelve; after twelve he is uncertain, the articulation and order becoming rapidly worse. He is conscious when he makes a mistake, but cannot correct himself, and ends in a hopeless muddle.

"In reading from a book, the words he pronounces have no relation to the print, either in length or sound; neither does he seem to understand written characters, as he will not attempt to answer a question written on a slate, though he will at once endeavour to respond when the same question is put to him orally. He, however, recognises figures from 1 to 9 when written, and is conscious when they are not placed in regular order. He cannot name any coins, but seems to have some idea of their relative value. He indicated on his fingers that sixpence was worth six pennies—not being able from sight to utter its name."

The patient was discharged from the hospital on April 6th, but was readmitted on April 16th, on account of his having had two fits on the morning of that day. The first occurred about 5.30 a.m., lasting about five minutes, during which time he was unconscious; the second, after an interval of an hour, was more severe, lasting about ten minutes. His wife said "his whole body was in a great tremor, and though his right limbs were free from the jerking, they were sometimes clenched tightly." The second fit left his speech rather worse than it was before, and his right limbs also became quite powerless.

On examination he was found to be quite unable to stand, or even to move the right leg to any appreciable extent. The right arm was also quite powerless. The right angle of the mouth was distinctly lower than the left; and the protruded tongue deviated considerably to

the right side. The sensibility of the right side of the body did not seem to be further impaired. His speech was rather more limited than when he went out. He could say "Yes" and "No," and "Very well" distinctly, but could not pronounce his name clearly. He could just manage to count from one to twenty, but made many mistakes after ten.

Three days later the increase of limb paralysis had passed off, and the patient was again able to walk about the ward dragging his right foot slightly; and he could raise his right hand as high as his mouth. Two weeks after admission, though he had gained further power in his right arm and leg, it was noticed that his speech was as bad as ever. He could name any simple numeral that was written and pointed out to him, and he could also correctly add columns of three or four figures; but he altogether failed to name individual letters of the alphabet, however plain or large they might be. He could recognise common objects, such as a dog, a fowl, or a tree, in an engraving, and point out any one of them when asked to do so. But he could not volunteer the name even of the most familiar object to which he pointed.

May 8th.—Asked successively to name large, separate, printed capitals O, K, and G, from sight, on each occasion he said "P," and on D being pointed to, he called it "M"—though he repeated the name of each of these letters, without a moment's hesitation, after hearing it pronounced. Although there is this inability to name letters, words, or objects from sight, the patient now seems to understand simple sentences written or printed; thus, when the sentence "Have you a wife?" was written on a slate, it was perfectly evident that he understood the writing. His condition, however, in this respect seems to vary from time to time. In the sentences the meaning of which he comprehends, he is still quite unable to pronounce the individual words from sight, though after hearing them uttered he can articulate them at once, more or less distinctly.

Two days later he was observed reading something in the newspaper, and on being asked if he understood it (the report from a police court of a case of poisoning), he at once said he did, and unmistakably indicated by his gestures that this was true. With his left hand he could write his own name after a copy, but not easily without, and sometimes not at all. A less familiar word he did not even attempt to write from the sound, even when it had been distinctly heard and comprehended.

Discharged on May 14th, and sent to the Convalescent Home at Eastbourne.

It will be observed that this patient's state in the early part of the month of May was distinctly different from what it had been in the early part of April, previous to the occurrence of the two fits on April 16th. At the later date, whilst the patient's utterance in repeating words which he had heard had become more distinct, he could not even emit an unintelligible jargon in attempting to read. At the same time he had become able to understand what he read, though he still could not name a single letter at sight, nor could he write a single word from dictation—both these processes requiring for their performance the proper relation between the visual and the auditory word-centres, and therefore the integrity of the commissures by which they are united. That half of the commissure which conveys stimuli from the visual to the auditory word-centre (as in reading aloud), seems to have been more extensively damaged after the two fits than it was before (Fig. 1).

His speech defects had, in fact, now taken the form which they subsequently retained throughout the remainder of his life; these were (1) the inability to read aloud or name at sight, together with the inability to write any thing from dictation, which, in the absence of "word-deafness" or "word-blindness" implied a damage to the commissures between the auditory and the visual word-centres; (2) the almost complete loss of voluntary speech,

whilst imitative speech was retained, implying a lowered functional activity of the auditory word-centre; and (3) the mere aphemic difficulty of utterance, implying some damage to the outgoing fibres between the third frontal

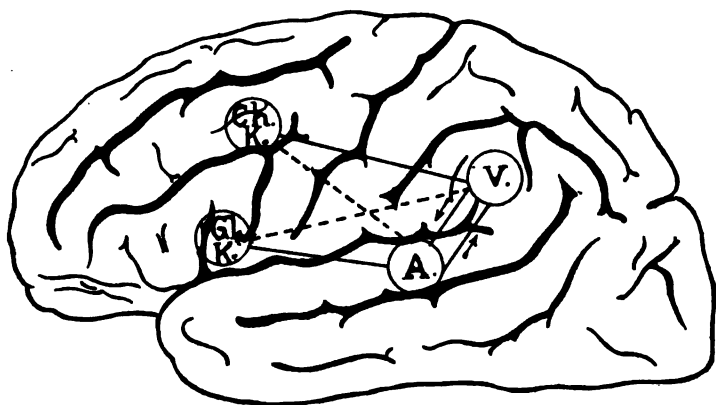


FIG. 1.—Diagram showing the approximate sites of the four word-centres and the commissures by which they are connected. The two broken lines indicate occasional rather than customary routes for stimuli.

convolution and the bulbar motor centres for speech, though it might possibly have been due to the lowered activity of the auditory word-centre.

While his speech defects preserved a constant character through so many subsequent years, evidence of the extension of the original damage done to the patient's brain was furnished by the recurrence of fits from time to time, and by the increase, especially during the last two years of his life, in the amount of paralysis of the right limbs and in the degree of right-sided hemianæsthesia.

It will, I think, conduce to clearness if we deal first of all with the progress of the case in reference to the fits and the hemiplegia; reserving for subsequent consideration the study of the patient's mental condition and speech defects.

CONCERNING THE FITS, HEMIPLEGIA, AND HEMIANÆSTHESIA.

1879.

January 21st—February 6th.—Has had no distinct fit since he was in hospital last year. The paralysis of the right side of the face remains about the same. The tongue is now protruded in the middle line. There is paresis of the right limbs, but no distinct paralysis. He can flex and extend his fingers, though very slowly. Dynamometer, right 25, left 85. He can also flex and extend the wrist, though with difficulty. He can flex and extend the elbow with about half the force that he exerts on the left side. He moves the shoulder-joint quite freely, but the movements are less powerful than on the other side. In walking he drags the right foot along the ground, and circumducts the leg. He can flex the hip-joints and the knee-joints with almost equal force on the two sides—the force on the right side being slightly less. He can also move the right ankle-joint freely. Over the right arm the patient can feel the light touch of a finger as far down as the wrist, but below that, over the back and front of the hand, he can only appreciate a pin's point. Sensibility in the right leg is fairly good.

1882.

July 15th.—Has had no fits for twelve months. Motor weakness on the right side remains about the same. Sensibility to touch, pain, heat and cold are distinctly impaired all over the body on the right side, including the face. He is short-sighted, but vision is otherwise normal. He hears a watch eight inches from each ear.

October 28th.—He is in much the same condition. He has had five fits since last seen, of a one-sided character.

1883.

April 20th.—Patient had four or five fits last week.

About the end of May he had two fits, each of which, according to his wife's statement, only lasted two or three

minutes. She says that during the fits he sometimes does not lose consciousness ; that the right leg is drawn up, but that there are no actual convulsions.

1886.

November 1st.—He can move the fingers of the right hand, but slowly. He can bend both his elbows perfectly, and can lift his arms at the shoulder-joints, but the right not quite so well as the left. He can walk well, but the right leg is stiff in its movements as compared with the left. There is no rigidity in the joints of either limb.

Plantar and patellar reflexes exaggerated on the right side ; ankle-clonus absent. He can feel the slightest touch on both sides of the body, but he himself intimates that he can feel it more distinctly on the left side. Sensation to pain is distinctly blunted on the right side, a pin's point not being felt as a prick, unless it is pushed in with some force. Sensation to heat and cold is also impaired on the right side, patient not being able to discriminate slight differences of heat and cold, and the application had to be made with some force for him to distinguish heat from cold. These defects of sensibility exist over the right side of the face as they did in 1882. He now hears a watch only within about half an inch of either ear. The paralysis of the right side of the face is a trifle less, as there is now no distinct asymmetry of face, and he is able to whistle. The tongue is protruded in the middle line. The field of vision is less extensive on the right side.

1894.

November 23rd.—Paresis of right arm and leg, with slight rigidity of all joints. He can only just grip with the right hand ; right 20, left 75. Flexion of the right knee is easily prevented. In walking there is obvious weakness of the right leg, which is brought forward with some difficulty, the toes dragging. There is slight right

facial paralysis. The tongue is protruded markedly to the right. Tactile sensibility is completely lost over the right hand and foot ; above it is diminished. There is partial analgesia all over the right side of the head and neck, limbs, and trunk. Sensibility to heat and cold is lost all over the right side, except in the right side of the tongue, where it is only diminished. He complains of pains of a dull, aching, and intermittent character, not increased on movement, in the right side of the head, the right arm and leg, and in the right side of the trunk. (He seems to have come into the hospital this time principally on account of these pains. They soon diminished, however, under the influence of potassium iodide and phenacetine ; his power of walking also slightly improved.)

Hearing.—On right side hears watch at one and a half inches ; on left side only with contact.

Vision.—Has to wear glasses ; without these he can only distinguish fingers with the right eye at a distance of about six inches. Sight much better with the left eye.

1895.

February 25th.—One week ago patient had a very severe fit,¹ after which he remained unconscious for three hours, and was unable to speak for some hours after recovering consciousness. He then had pains in the right arm, trunk, and leg, and very slightly in his head. He found himself quite unable to move the arm or the leg, and remained in bed till he was brought to the hospital in the ambulance.

On admission the following notes were taken concerning his condition. He still complains of pains in the right side of the body, and slightly in the head. He cannot raise the right foot from the bed, and the arm only to a very slight degree. He has great difficulty in moving the fingers. Tactile sensibility is lost below the elbow of the right arm, and is very much diminished over the rest of

¹ The last previous fits of which there is any record being in 1883.

the right side of the body. Sensibility to painful impressions is also very considerably blunted over the whole of the right side, but is absent nowhere. The loss of sensibility in the leg is most marked distally, being almost complete below the knee. There is considerable rigidity of the right arm and leg.

The knee-jerk is exaggerated, and ankle-clonus is present on the right side; left side normal.

March 3rd.—Under treatment the pains have disappeared, the rigidities are passing off, and he is recovering more power in the right arm and leg. He is able to walk about the ward with some difficulty.

14th.—Power of arm has greatly improved, and of leg also. There is almost complete loss of muscular sense in the right arm.

Readmitted December 4th, 1895. On November 24th he had another fit in which he fell down completely unconscious. The twitching was confined to the right side of the body. Twenty minutes later he had a second fit of the same character. In the course of a few hours he regained consciousness, and then found himself considerably weaker on the right side than he had been before. Since then he has kept to his bed, up to the day of admission, when the following notes were made:—The whole of the right side is partially paralysed. He has considerable power of movement at the shoulder and elbow, but all the right fingers are completely paralysed, there being no power of flexion or of extension. The right leg is much weaker than the left. The knee-jerks are exaggerated on both sides.

December 12th.—Tactile sensibility is diminished all over the right side of the face, and he is not able to localise the points where he is touched. There is loss of sensation to touch and pain all over the right arm; that of pain is completely lost, while touch is only diminished. Sensation to heat and cold is also completely lost in the right arm, but not completely in the face—cold being much more readily distinguished than heat. The

muscular sense, as far as it can be tested, seems to be almost completely lost. There is considerable rigidity of the right arm with exaggerated wrist-jerk, also rigidity of the right leg with exaggerated reflexes. Sensibility to pain, heat, and cold are lost in the right leg, and sensibility to touch is very deficient. Muscular sense also seems to be lost. Discharged January 4th, 1896.

CONCERNING THE MENTAL CONDITION AND SPEECH DEFECTS.

1879.

Patient is quite rational, and able to play games such as draughts.

The Auditory Word-centre with its Afferent and Efferent Fibres.

He readily understands any question put to him. He can repeat short words correctly, but there is often an aphemic difficulty in utterance. Thus, he cannot properly pronounce words beginning with the labials, but those beginning with gutturals he pronounces much better. He can count quite correctly up to twenty, but beyond this gets rather confused with the repetition of the first word. When started, he can say the alphabet correctly as far as "i," after that very badly. His spontaneous speech is very limited; but one day before returning to the hospital he said, "Never mind, they may come to-morrow;" and on another occasion, "Mrs. Foster will come to-morrow." Otherwise his speech has been limited, as at present, to "Yes" and "No," "No, it isn't," "Good morning," and some simple expressions of this kind.

The Visual Word-centre with its Afferent and Efferent Fibres.

He understands any written question. He reads books and newspapers much. He says he understands what he reads, and there is every reason to believe that this is

true. He could detect mistakes in his wife's accounts, but could never explain the error to her. He can add two rows of simple figures. Subtraction he finds more difficult, but can get a simple sum right after a short time. Over a simple sum in multiplication he requires some prompting before he gets it right. He can copy writing well with his left hand; but he cannot transfer from printed to written characters, or *vice versa*. Can write nothing spontaneously, not even his own name.

Commissures between the Auditory and the Visual Word-centres.

a. He cannot read aloud at all either from print or writing. He cannot read aloud the simplest words or even name single letters, except that occasionally he can name *a* or *o* correctly. (His disability in this respect is always most striking when contrasted with the rapidity with which he pronounces the same word or letter as soon as he hears it uttered.)

b. He cannot write anything from dictation; but if a short word is spelt for him he can sometimes write the letter just named after a long time and with much difficulty. (His inability to write a single word from dictation always contrasts notably with the readiness with which he will copy the same word as soon as it is written.) Simple numerals, such as 7, he can write from dictation; 12 he finds more difficult, making it 31, then 10, and shortly afterwards 12. He puts on the 5 at the right place to make it 125.

1886.¹

He appears to be a man of ordinary intelligence. He is well-behaved, and seems perfectly happy and good-tempered. He takes a great interest in what goes on

¹ He was examined as to his mental condition and speech when he was in the hospital in 1882, and also in 1883, when his condition was found to be substantially the same as it was in 1879, except that he could not on either occasion name a single letter at sight—not even A—and in the former year

about him, and busies himself reading the newspaper and playing draughts—a game he plays remarkably well. His memory is fairly good, but since his admission he has given his age differently several times, and he also varies as to the date at which he was in the hospital last. He, however, recognised the ward and bed in which he was when first admitted in 1878.

The Auditory Word-centre with its Afferent and Efferent Fibres.

He comprehends everything that is said to him, and listens to the conversation of the other patients. He can distinguish one tune from another perfectly; *e. g.* when asked whether he knew "Home, sweet home," he said "Yes," and he did recognise it at once from other tunes. Can repeat short words correctly, but makes a hopeless muddle with some long ones like "constitution." When started, he can say the letters of the alphabet pretty correctly as far as *n*, afterwards with mistakes and omissions. He can name all the days of the week. Cannot name the day of the week without beginning "Sunday, Monday, &c.," and then stopping at the right day. He can say the names of the months as far as July, but cannot recall the rest. His spontaneous speech, apart from associational trains, is extremely limited. He says "Yes" and "No," and "Very well" when asked how he is. If asked his name he can say "Andrews" correctly, but not "Thomas;" and is unable to say where he lives. He can also say "beer," "bacca," and a few words such as "tea" and "dinner." When asked how old he is, he says "forty," and then counts on his fingers 1, 2, 3, 4, 5, 6, 7, 8, 9, and stopping nods his head—his age being forty-nine. He has not used a verb or adjective since admission, except to say "No, it ain't."

it was found that he could write numerals spontaneously with the aid of an associational nexus. Thus he wrote them from 13 to 70 correctly, except that on each occasion he missed the 9 and wrote 7 instead—writing, "15, 16, 17, 18, 17, 20," and so with each subsequent decade.

The Visual Word-centre with its Afferent and Efferent Fibres.

He understands all that he reads, and passes most of his time reading a newspaper or a book. He is able to show by signs what he is reading about. He can copy any numeral, letter, or word with his left hand. He will not, however, attempt transfer copying. He can write spontaneously and correctly the numerals from 1 to 10. He cannot write a single letter or word spontaneously.

Commissures between the Auditory and the Visual Word-centres.

a. He can sometimes name at sight certain familiar objects having short names, such as "pen," "book," "pin," these being about his only achievements in this direction. Cannot name numerals except by counting up to them from the beginning. He cannot name any letters except occasionally *a*, *b*, or *c*. The same applies to words, though very rarely he can name a short word correctly, *e. g.* "so," "goat," "dog." He is quite unable to read aloud even the shortest sentence.

b. He can often write numerals and simple numbers correctly from dictation, *e. g.* 7, 12, 78—but not numbers with three figures. For 281 he wrote 1249. As a rule he cannot write a single letter from dictation. Rarely, however, he can write *a* or *b*, but no other letter. He was never able to write even the shortest word from dictation, though he will immediately copy any word that has been written and placed before him.

1894.

The Auditory Word-centre with its Afferent and Efferent Fibres.

Understands all that is said to him. He can repeat all simple common words, but uncommon polysyllabic words he is unsuccessful with, producing mostly a mere jumble

of sounds. He can repeat the numerals up to 20; and can say the alphabet when started as far as *m*, but cannot go beyond without missing many letters. Can name the days of the week, but often omits Sunday. His spontaneous speech is limited to his name (he cannot give his address), together with short affirmative or negative answers, and occasionally very short phrases such as "Never mind."

The Visual Word-centre with its Afferent and Efferent Fibres.

He understands what he reads. Recognises all words, letters, numerals, common objects, and pictures. He can copy numerals, letters, and words quite well, with the left hand. He can go on writing numerals when the first one is given, but letters and days of the week cannot be written in the same way. He cannot write a single letter or word spontaneously.

Commissures between the Auditory and the Visual Word-centre.

a. Cannot name at sight words, letters, numerals, or common objects.

b. He can write simple numerals from dictation. Cannot write a single letter or word from dictation.

No systematic examination of the patient's mental condition and speech defects was recorded after this date, but when admitted to the hospital on February 25th, 1895, after the very severe fit with increased paralysis in the limbs, there is a note in these terms:—"His mental condition and speech defects remain the same as when he was in before, except that he is rather less demonstrative and cheerful, being somewhat despondent over his relapse." Again, when he came to the hospital after two other severe fits on December 4th, 1895, there is the following note:—"His speech remains in the same condition

as on previous occasions. There has been no alteration in his condition except increased weakness in the limbs on the right side." Later, on December 16th, there also appears the following note:—"Patient can name some common objects, but cannot put together any sentence. He can count readily up to twenty. He can begin the alphabet, which he had not been able to do on previous occasions. Can say the days of the week. Cannot repeat a sentence after one with more than two or three words, but can repeat single words fairly readily. Can copy words with the left hand, but cannot write words from dictation or spontaneously. When shown a boot to-day, he wrote 'boot' on a paper fairly well. He can read the newspaper and understand it, but cannot read aloud."

He was discharged from the hospital on January 4th, 1896. He came to the ward to see me on two occasions subsequently, the last time being about the beginning of March, when I demonstrated his condition to the students, and found it to have undergone no appreciable change.

He died on March 29th, and as to the manner of his death I will give a few particulars, kindly furnished to me by Mr. Naunton Ruck, of Ealing, who was called to see him. He writes:—"I can add little to his wife's account of his illness.¹ He was apparently in his usual health on the morning of the 28th, and was standing when the seizure came on. He fell down, and must have rapidly lost consciousness, for I saw him at 11 o'clock, soon after the seizure, and he was then quite insensible. He was very much convulsed, and the convulsions lasted for two hours. He became and continued comatose till his death at 9.30 p.m. on the following (Sunday) evening. He was unable to swallow, and at no time did he show any signs of rallying."

Permission having been obtained to examine the head, my then house physician, Dr. Harold C. Way, was good enough to go to Ealing and remove the brain. This was

That is some particulars which she had sent to me.

done about thirty-eight hours after death. There was nothing unnatural about the skull-cap or the dura mater, but on the left side of the brain there was what appeared to be a large pseudo-cyst. The organ was subsequently handed over to Dr. J. S. Risien Russell for careful preservation and subsequent report. This service he was kind enough to render, and I am indebted to him for the following careful report, the accuracy of which I have been able to verify.

Report on the Brain of Thomas Andrews.

"The brain showed evidence of old softening resulting in complete atrophy of the left hemisphere throughout the whole of the area of distribution of the middle cerebral artery, with the exception of the area usually supplied by the first cortical branch of this artery, which area was intact, and thus Broca's convolution had escaped softening and subsequent atrophy. There was, however, well-marked extension of the atrophy into the posterior portion of the second frontal convolution, corresponding to the area of distribution of a branch of the second branch of the middle cerebral which supplies this area.

"An unusual amount of the upper part of the ascending frontal and parietal convolutions had escaped softening and subsequent atrophy, viz. $4\frac{1}{2}$ cm. of the former, and $3\frac{1}{2}$ cm. of the latter, which portions presented a healthy and unaltered appearance. The total lengths of these convolutions on the right side were, ascending frontal $10\frac{3}{4}$ cm., and ascending parietal 10 cm.

"The angular and marginal convolutions were destroyed; but almost the whole of the superior parietal convolution had escaped, the width of the intact area of cortex in this region amounting to a little more than 3 cm.

"The superior and inferior occipital convolutions were intact, but the atrophy had extended to an unusual degree into the middle occipital convolution, 3 cm. of this convolution being intact, however.

"The whole of the superior temporo-sphenoidal convolution was destroyed with the exception of the anterior one third, $4\frac{1}{2}$ cm. in length. Of the middle temporo-sphenoidal convolution, the anterior 5 cm. were perfectly intact; but posteriorly only a narrow portion of the inferior part of this convolution remained, and that was in a discoloured and degenerated condition.

"The whole of the atrophic area that has been described (from which all trace of brain tissue had completely disappeared) was occupied by a large pseudo-cyst which was continuous with the lateral ventricle, the fluid with which it was filled being shut in by the slightly thickened pia arachnoid" (Plate XV, fig. 2).

"On examination of the thrombosed branches of the left middle cerebral it was impossible to ascertain in which of these the process had commenced. The trunk of the vessel and all its branches had now become much attenuated. When the brain had been hardened and the membranes completely removed from the left hemisphere a great gap was found in the region indicated, 11 cm. in length by $3\frac{1}{2}$ cm. broad, and 4 cm. in maximum depth (Plate XV, fig. 3). The upper part of the descending and nearly the whole of the posterior cornu, together with the posterior half of the body of the lateral ventricle, were opened up. Much of the posterior segment of the internal capsule together with the greater part of the thalamus had disappeared, the latter being represented only by a small rounded portion, having rather less than one third of the bulk of a normal thalamus. Anteriorly the atrophy had extended into the white substance up to the corpus striatum, which was also much diminished in size. All other portions of the cortex of the left hemisphere beyond the region above defined presented a perfectly healthy appearance. The left half of the pons was slightly flatter than the right, whilst the left pyramid was very distinctly smaller than its fellow. The lateral lobes of the cerebellum showed no inequality.

"The right middle cerebral artery was occluded by a

recent thrombus. There was practically no evidence of softening of the cortex of this hemisphere (nor were there lesions of any other kind), but there was extensive softening of the basal ganglia and internal capsule, probably accentuated by post-mortem changes."

Comments.—The details furnished concerning the first attack in December, 1877, were not sufficient to enable one to decide positively whether it was caused by thrombosis or by hæmorrhage; and, curiously enough, the sudden onset and rapid termination of the final apoplectic attack would also rather have suggested hæmorrhage than thrombosis, especially to those who are not fully aware of the extreme difficulty that often exists in deciding between these two causes of an apoplectic or simple hemiplegic attack.

It seems clear that in the first attack of 1877, there could not have been a complete occlusion of the middle cerebral artery. The transitory nature of the complete paralysis, as well as some of the other symptoms, were unfavorable to this view. It was, moreover, negatived by the post-mortem revelation that the third frontal convolution was intact. The occlusion of the vessel was, therefore, evidently beyond the point whence the first cortical branch is given off, which is the nutrient artery of this convolution.¹

It is even difficult to suppose that at first the whole of the other cortical branches of the middle cerebral were involved. Had this been so it seems clear that there must have been a more than usually free anastomosis between the terminal ramifications of these branches and those of the corresponding anterior and posterior cerebral arteries. That there was such a free anastomosis

¹ In the 'Transactions' of this Society for 1884, Dr. Sharkey has recorded an interesting case where death occurred seven years after a complete thrombosis of the right middle cerebral artery, in which the third frontal and also a part of the orbital convolutions had undergone softening and atrophy. The lesion in this case being on the right side, speech was "but slightly affected."

between the second and third cortical branches of the middle cerebral artery seems evidenced by the unusually small amount of the customary territory of these vessels which underwent softening and atrophy. The same kind of evidence does not, however, tell in this direction for the fourth branch, in the territory of which there was found a full amount of atrophy.

It is the clinical evidence that makes it improbable that the whole of the territory of this fourth branch was at first cut off from its blood-supply. Had this been the case, word-deafness as well as word-blindness would have been produced. But no word-deafness seems ever to have existed in the early stages of this man's illness; and though there was word-blindness at first, this had completely cleared up early in May—that is about five months after the onset of the hemiplegia.

At this latter date the speech defects assumed the form that they maintained during the following eighteen years, and were of such a kind as (apart perhaps from the mere aphemic difficulty in articulation) to imply a lowered condition of functional activity in the auditory word-centre (*i. e.* in the posterior half of the upper temporal convolution), a fairly healthy condition of the visual word-centre (*i. e.* of the supra-marginal and angular convolutions), together with great damage to the important commissures connecting these centres with one another (Fig. 1).

Though this was in all probability the condition existing for an indefinite period from May, 1878, onwards, the results of the post-mortem examination showed that a progressive destruction must have taken place in these auditory and visual word-centres. This being so, one of the most remarkable features of this case will be found to be the unvarying character of the speech defect, notwithstanding the progressive destruction of such centres.

A similarly progressive destruction of brain tissue must have extended even into parts of the hemisphere beyond the field of distribution of the terminal ramifications of

this fourth cortical branch of the middle cerebral—that is into the contiguous territory of some of the anastomosing branches of the posterior cerebral. This is shown by the amount of atrophy that had taken place in the white substance of the hemisphere, in the posterior part of the internal capsule, and in the thalamus.

The increased sensory and motor paralysis that occurred during the last two years of the patient's life was almost certainly due to the destruction of the posterior part of the internal capsule. This would account also for the almost complete loss of muscular sense reported to exist (in 1895) in both limbs, when the motor paralysis was still very incomplete, and for the loss of tactile and other modes of sensibility in the foot and lower part of the leg, whilst the foot and leg areas of the convolutions were intact.

In regard to the clinical indications as to the actual nature of the speech defects, not much remains to be said after the detailed and classified notes that have been given. The deductions that seem warranted are these :

(1) That a marked degree of amnesia existed, due to an impaired functional activity of the auditory word-centre.

That the activity of this centre was merely impaired, and not abolished, was shown by the fact that the patient understood speech perfectly, and that his own imitative speech was preserved, although spontaneous speech was almost lost.

(2) That the activity of the visual word-centre did not seem to be appreciably impaired.

This was shown by the complete absence of alexia, and by the patient's ability to copy writing.

(3) That there was more or less complete destruction of the double commissure existing between the auditory and the visual word-centres.

That the *visuo-auditory commissure* was interrupted seemed shown by the fact that the patient could not read aloud, or name at sight, though he could understand what he read, and recognise all common objects.¹

¹ His only achievements in this direction that have been recorded are these.

In patients showing the degree of amnesia present in this case it has often been found that the auditory word-centre will respond to a strong stimulus coming to it from the visual word-centre, as is shown by the fact that these patients are able to read aloud quite readily though their power of spontaneous speech is almost lost. The destruction of the visuo-auditory commissure, however, made this achievement impossible in the case of Thomas Andrews.

That the *audito-visual commissure* was interrupted seemed shown by the fact that he could not write a single word from dictation,¹ though he could copy any such word at once; and that he could even, on the last occasion of his being in the hospital, write "boot" on being shown that article. That he could write numerals from dictation and also name them at sight is difficult to understand. But a greater power of understanding, of naming, or of writing numerals, as compared with letters of the alphabet, has been very commonly met with in the investigation of speech defects.

The patient's greatest power of writing spontaneously

In 1879 he could sometimes name the letters *a* and *o* correctly; in 1886 he sometimes named *a*, *b*, or *c*, and once or more named the short words "so," "goat," and "dog;" whilst in this same year, when, as I have noted, he was at his best, he also succeeded in naming "pen," "book," and "pin," when these common objects with short names were shown to him. There is, of course, the possibility that he may have been able to succeed to this extent by stimulating Broca's convolution direct from the visual word-centre, thus avoiding the necessity for the passage of the stimulus by way of the visuo-auditory commissure to the auditory word-centre and thence on to Broca's convolution. This more circuitous route is, as I believe, the rule in reading aloud and in naming at sight. The majority of writers on speech defects seem, however, to think that the more direct route is habitually followed by the stimuli concerned in the execution of these acts—a view that is not at all borne out by the case now under consideration.

¹ His only achievements in this direction that have been recorded are these. In 1879 he could sometimes write single letters from dictation, and generally single numerals. In 1886 he could write numbers with two figures, but not those with three, though he could rarely write letters from dictation other than *a* or *b*. In 1894 he could still write numerals, but not a single letter from dictation.

was shown in 1878, when he sometimes succeeded in writing his own name more or less correctly. This ability did not persist; but subsequently in 1883 and also in 1886 he was found able to write numerals in series spontaneously, and almost correctly. His inability to do anything more in the way of writing spontaneously was, I think, due to two causes: (1) the low activity of his auditory word-centre, which also prevented his spontaneous speech (activity in this centre being for most persons the necessary first stage for the initiation of spontaneous writing); and (2) the fact of the damage to the audito-visual commissure, which would have prevented the passage of stimuli across to related portions of the visual word-centre. His ability occasionally to write such a very familiar word as his own name or simple numerals spontaneously was, I think, due to the fact that in each of these cases the necessary images and stimuli may have been called up primarily in the visual centre, just as, on the occasion above referred to, the sight of a boot enabled him to revive the associated literal symbols, and write the word "boot."

(4) That he had an aphemic difficulty of utterance, rather more marked in the early years of his illness than it was later, which might have been due to a defect in the co-operative action of the weakened auditory word-centre with the kinæsthetic centre in Broca's convolution, though it is more commonly due to a defect in the outgoing channels leading from the latter centre.

How are the Clinical Peculiarities of the Speech Defects to be reconciled with the Post-mortem Record?

The difficulties in reconciling the persistent and often-verified clinical condition with the post-mortem record are extreme. As I have said, in the absence of word-deafness and word-blindness as initial symptoms, one cannot suppose that complete softening at first existed in the auditory and the visual word-centres.

The total atrophy in these regions found after a period of eighteen years seems, therefore, as above stated, irreconcilable with the fact shown by the clinical records that during this long series of years there had been no appreciable change in the speech defects. How, then, it must be asked, could this complete atrophy have occurred in the region of the auditory and the visual word-centres without manifesting itself clinically?

We must, I think, conclude that the destruction of these parts had been gradual. Had it been sudden, all present knowledge entitles us to conclude that it must have revealed itself by the occurrence of well-known symptoms, namely, word-deafness and word-blindness in combination. And assuming that the destruction had been gradual, we can only suppose that this process must have coincided with a gradual development in the functional activities of the corresponding convolutional regions of the right hemisphere.

We know next to nothing as to the relative degree of organisation of the right as compared with the left auditory and visual word-centres. *A priori* considerations would make it probable that corresponding auditory and visual word impressions impinging on each side of the brain of a person whose hearing and sight were good on both sides, ought to become associated with similar structural organisations in relation with these afferent impressions.

It is well known, however, that the executive functions in connection with these two centres are carried on in right-handed persons wholly, or in the main, by the left side of the brain; and this fact must, it would seem of necessity, entail an arrest in the development of the commissures between the auditory and the visual word-centres in the right hemisphere, answering to those by means of which these executive functions are carried out on the left side of the brain. The development of the right centres being thus arrested, their autonomy would necessarily be hindered.

In many cases where recovery from different kinds of

speech defects occurs (the nature of which I need not now particularise) we are compelled to assume that centres and commissures in the right hemisphere gradually undergo further organisation, so as to compensate for the destruction of corresponding structures in the left hemisphere.

It can only be supposed, therefore, that something similar must have occurred in the case of Thomas Andrews.

What is altogether novel and surprising, however, is that restoration should only have taken place in such a very imperfect manner—that imperceptibly, as it were, the imperfect functioning of the left hemisphere, and no more, should have been taken on by the right hemisphere. It may, perhaps, be supposed that the low, though unequal, activity which was alone attained in the right auditory and visual word-centres did not even suffice to develop the usual commissures between these two centres, and that thus the exact clinical pattern and combination of speech defects should have been preserved, even when the cerebral activity on which they were dependent had been gradually transferred from the left to the right cerebral hemisphere.

Whilst offering these mere suggestions concerning a very difficult problem, I fully realise how much they fall short of anything like an adequate explanation of the very puzzling riddle presented by this case.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, pp. 20, 25.)

DESCRIPTION OF PLATE XV.

On a Case of Amnesia and other Speech Defects of Eighteen Years
Duration, with Autopsy (H. CHARLTON BASTIAN).

Fig. 2. — Brain of Thomas Andrews before removal of the
membranes.

Fig. 3. — Brain of Thomas Andrews after removal of the
membranes.

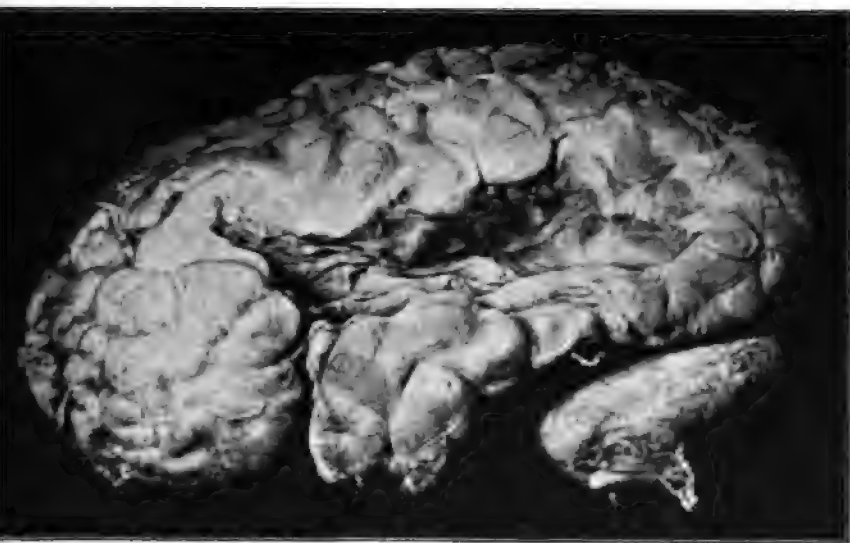


Fig. 2.



Fig. 3.



INFANTILE CEREBRAL DEGENERATION

WITH

SYMMETRICAL CHANGES AT THE MACULA

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INTRODUCTION.

UNDER the above title we purpose to describe a rare and fatal disease of infancy, which commencing in the early months of infant life by muscular enfeeblement associated with distinctive ophthalmoscopic appearances, progresses to almost complete paralysis, and terminates in the death of the patient about the end of the second year.

So far as we know the disease has been chiefly recognised by ophthalmic surgeons, their recorded observations dealing mainly with the ocular symptoms.

Mr. Waren Tay first drew attention to the subject in 1881, and the references to his papers, and to those of others who have recorded instances of the affection, will be found in a bibliography at the end of this paper.

No autopsy could be obtained in any of the recorded cases, with the exception of a case reported by Dr. Knapp. A description of the changes found in the brain in this case is given by Dr. B. Sachs, of New York, in a paper entitled "Arrested Cerebral Development," a reference to which will also be found in the bibliography on the subject. The spinal cord and eyes were not examined in the case in question.

Three cases of the disease have been under the observation of one of us (Kingdon), and careful clinical investigation of the patients has been supplemented by post-mortem examinations together with a microscopical examination of the central and peripheral nervous systems and eyes in two of the cases which have terminated fatally. We propose, therefore, to give an account of these cases in the first instance, then to allude to one kindly placed at our disposal by Dr. F. J. Smith, and finally to draw attention to the leading characteristics of the disease, our description being based on a study of the recorded cases in conjunction with those that we are about to call attention to.

CASES OBSERVED BY THE AUTHORS.

The patients were two brothers and a sister, born of strong and healthy parents, both of whom are German Jews. The parents are not related to each other, and no family history of any hereditary taint can be elicited.

The family consisted of seven children. The first, a boy born one year after marriage, was, according to the mother's statement, well developed at birth, but subsequently became weak in the back and limbs, and mentally apathetic; he wasted and died at two years of age in the Hull Infirmary, and was without doubt the subject of this complaint.

The second child, a girl, born ten months afterwards, is in good health at the present time.

The third child, a boy, born three and a half years afterwards (no miscarriages in the interval), was affected, and forms Case 1.

The fourth child, a boy, born April, 1892, is healthy.

The fifth child, a girl, born May 6th, 1893, was affected, and forms Case 2.

The sixth child, a boy, born November, 1894, is affected, and forms Case 3.

The seventh child, a boy, born November 21st, 1895, appeared healthy when examined on February 20th, 1896.¹

CASE 1.—An account of this patient with the result of the autopsy was read by one of us (Kingdon) before the Ophthalmological Society in 1892, and was published in the 'Transactions' of the Ophthalmological Society of the United Kingdom, vol. xii, p. 126. The following is a brief abstract :

Jacob R—, aged 8 months, was admitted to the Children's Hospital, Nottingham, on account of a general increasing weakness of the back and limbs (Fig. 1).

The mother stated that the patient was born at full term, and until the third month was reached, he appeared to have similar strength to other children of the same age. He was fed by the breast until his admission, and had always been well nourished. There had been no convulsions or other previous illness. From the age of three months there had been increasing enfeeblement of the muscles of the back and limbs, the most obvious symptom being an inability to hold up the head.

On July 1st, 1891, the child, being nine months old, was 17½ lbs. in weight, 26½ inches in height, and had a well-shaped head 17½ inches in circumference. There were no external congenital defects; the body was well covered

¹ Since this paper was submitted to the Society, unmistakable evidence of the disease has been detected in the seventh child of the family. The sixth child (Case 3) died on May 6th, 1896; permission to examine the head was obtained, and a microscopical examination of the brain, cerebellum, pons, medulla, spinal cord, and eyes has since been made.

with fat; the muscles felt flabby, but any wasting was masked by the subcutaneous fat. The limbs moved feebly,



FIG. 1.—A photograph of the boy an abstract of whose case has been included in this paper as Case 1. The photograph was taken in October, 1891, and shows little beyond the cerebral expression of face, and the weakness of the neck muscles as evinced by the falling back of the head.

and though objects were grasped when placed in the hands, they were soon dropped. He was unable to turn on to either side when lying on his back. The knee-jerks were well marked. There was no rigidity of the limbs, but occasionally there were some short spasmodic contractions. The child was apathetic, and lay quietly in bed. Hearing was acute, with great sensitiveness to sudden noises. The eyes on external examination appeared normal, but no notice was taken of objects held in front of them; and on ophthalmoscopic examination symmetrical changes

at the macula were discovered. In this region there was a whitish-grey patch about twice the size of the optic disc, only slightly raised above the general surface of the retina, with a dark cherry-red spot in its centre. The thoracic and abdominal viscera were healthy, the appetite good, the bowels regular, the urine natural, and the temperature normal.

The child remained in much the same condition until October 16th, though more feeble and quite blind as a result of optic atrophy. He had increased 1 lb. in weight. Two days afterwards he became pale and shrunken, and gradually unconscious, with frequent moaning and low rhythmical movements of the eyes from side to side; the pupils were equal, moderately dilated, and inactive to light. There were no definite convulsions. The temperature remained normal, and there were no signs of disease in the chest. The condition resembled the attacks which occur during the later stages of general paralysis of the insane.

The patient became more lethargic, and died on October 20th, 1891.

Autopsy (eighteen hours after death).—The brain with the upper portion of the spinal cord and both eyes, were removed and placed in Müller's fluid. The thorax and abdomen were not examined. The posterior fontanelle was closed, the anterior fontanelle nearly so. There was no marked excess of cerebro-spinal fluid. The arachnoid and pia mater were neither thickened nor adherent, and there was no evidence of meningitis. The sulci over the whole surface of the brain were slightly wider than normal, but there was no irregularity in the arrangement of the primary fissures or convolutions. The distribution of the superficial blood-vessels was normal. The brain weighed 38 ounces.

Microscopical examination.—The microscopic examination of the brain and spinal cord was not so thorough as that which has been carried out by one of us (Russell) in the second case. Sections taken from the different cortical areas revealed very similar changes in all, viz. that the different layers of cells were not so readily distinguished

as is usual, the most marked alteration appearing in the large pyramidal cells, which had an oval or rounded outline. The protoplasm was vacuolated, and mostly collected into an irregular shrunken mass around the nucleus. The same changes were present in the small pyramidal cells. The basal ganglia were not examined.

Sections of the spinal cord at the level of the second cervical vertebra revealed well-marked descending degeneration.

Ocular changes.—The eyes were embedded in celloidin, and the changes found were similar in both. The retina was folded at the macular region; the outer molecular layer of the retina in this area was spaced out and enlarged, but the other elements of the retina seemed healthy. The optic nerve was atrophied. The choroid was normal.

Treatment.—For the first two months after admission the child was given Pulv. Hydrarg. \bar{c} . Cret. gr. $\frac{1}{4}$ twice daily. After this time no medicinal remedies were employed beyond such as were necessary to check any temporary alimentary disorder. Great attention, however, was devoted to the careful and regular feeding of the child, and to providing that its general surroundings should be as favorable as possible.

CASE 2.—Bluma R—, sister of the above, born May 6th, 1893, was on August 5th, when three months old, brought to one of us (Kingdon) to have the eyes examined, as the mother thought her child was failing in the same way as the others who had died. On examination some weakness of the back and neck was observed, and the mother stated that the child “started” occasionally while asleep. The condition of the macula lutea and optic papilla was normal.

When five months old another examination was made. The muscular enfeeblement had advanced, and the child paid but little attention to objects placed in its hands or held before its eyes. Ophthalmoscopically there was a suspicious haze at each macula.

December 26th, 1893.—The child being now seven and a half months of age, was again seen, and admitted to the hospital, where it remained until its death. The general appearance, nutrition, and muscular weakness corresponded to that of the first case; but she was more fretful and less apathetic than her brother. Ophthalmoscopic examination revealed the typical changes in the fundus of each eye, similar to those met with in the boy. Hearing was acute, and the sense of taste preserved. The muscles at the back of the neck and intra-scapular region were obviously atrophied. The electrical reactions of the muscles were not tested. Cutaneous sensibility was apparently normal.

Her weight was 15 lbs. The appetite was good, the bowels regular, and there was no sickness. The heart, lungs, urine, and temperature were normal.

During the first fortnight after admission, the patient gained $1\frac{1}{2}$ lbs. in weight, and on January 25th, 1894, was shown at a meeting of the Ophthalmological Society in London. After this temporary increase in weight no further improvement occurred; on May 2nd she still only weighed 16 lbs., and the subcutaneous fat, as well as the muscles generally, had wasted. Rigidity of the latter was now evident, the head was retracted, the left forearm pronated and partially flexed, with flexion of the fingers and thumb of the left hand, both the thighs were adducted, the legs extended, and the great toes extended at the metatarso-phalangeal and flexed at the inter-phalangeal joint.

During June the general aspect of the patient grew worse, the wasting and rigidity of the body increased, and the face wore a greater cerebral expression; the appetite remained good, and the temperature normal.

During July and August there were occasional attacks of vomiting and diarrhoea, and food was given with difficulty. The rigidity of the lower limbs was extreme, so that they could not be bent. The thighs were flexed on the abdomen, rotated inwards and adducted until the knees

crossed each other (Fig. 2). There was partial backward dislocation of the knee-joint from hyperextension of the



FIG. 2.—A photograph of the inferior extremities of the same case as that represented in Fig. 3, intended to illustrate the marked adductor spasm which was present at an advanced stage of the disease.

legs (Fig. 3). Both upper arms were rotated inwards, and the forearms pronated (Fig. 3). The child evidently suffered much pain as the rigidity increased.

In September the weight was 11 lbs. 10 oz. There was total amaurosis, the appearances at the macula had undergone no change, but optic atrophy was distinct.

On November 11th the weight had fallen to 10 lbs. 4 oz.; nevertheless the child lingered until December 26th, 1894, when it died quietly from exhaustion at the age of 1 year and 8 months, the weight of the body being only 8 lbs. 15 oz.

The history of this case differs from that of the first, in that the boy died before the emaciation and secondary contractions of the muscles had taken place.

Autopsy (an hour and a half after death).—Both fontanelles were closed. On removing the calvaria the



FIG. 3.—This photograph of the girl whose case we have gone into more fully than the former, was taken on September 12th, 1894, and illustrates the characteristic features of an advanced stage of the disease. The child was emaciated, quite unable to sit up, and the different segments of its limbs variously distorted by the spasmodic overaction of muscles, the overextension at the right knee being particularly well shown in the photograph. The cerebral expression is still more marked in this case than in the former.

arachnoid and pia mater were seen to be normal in appearance; they were non-adherent to the brain, and there were no signs of meningitis. The same remarks apply to the membranes of the cord. There was no excess of cerebro-spinal fluid. The distribution of the main vessels at the base and on the upper surface of the brain was normal. The convolutions, fissures, and sulci were well marked, and the latter appeared somewhat wider than normal, but there was no very evident atrophy of the convolutions. The brain weighed 1 lb. 7 oz.; the general consistency was not noticeably altered, and there was no marked vascularity. Portions of the brain, spinal cord, and the sciatic nerve and both eyes were removed and at once placed in Müller's fluid,

which was subsequently changed at regular intervals. The abdominal and thoracic viscera appeared normal to the naked eye.

Microscopical examination.—When sufficiently hardened in Müller's fluid, some portions of the central nervous system and sciatic nerve were prepared for cutting in celloidin in the usual way, and the sections thus obtained were subjected to the influence of various stains, the most important of which were Pal's modification of Weigert's hæmatoxylin stain, picro-carmin, nigrosin, &c. Other portions of the central nervous system and sciatic nerve were cut into thin pieces, after removal from the Müller's fluid, and then placed in Marchi's solution, which consisted of one part of a 1 per cent. solution of osmic acid and two parts of Müller's fluid. The specimens were kept in this fluid, on an average, for about a week, the exact time depending on the size of the particular specimen; and the fluid was changed from time to time as occasion required. On removal from this solution the specimens were prepared in the usual way for cutting in celloidin, and the sections obtained were mounted for examination, no further stain being required as the staining is combined with the process of hardening in Marchi's solution.

In the case of the cerebral and cerebellar cortex, still another method of preparation was adopted: small thin portions of these tissues were transferred from Müller's fluid to Marchi's solution for thirty-six hours, and were then placed in a 0.75 per cent. solution of nitrate of silver for forty-eight hours. The specimens were prepared for cutting in celloidin, and mounted as in the case of those prepared by the Marchi method.

In describing the changes met with on microscopical examination of the various regions, it will, we think, be best to describe the changes as met with in any given part, as revealed by the various methods of staining, rather than to describe under each method of staining the defects met with in this or that region.

Cortex cerebri.—Portions removed from the following

regions were examined after preparation by the various methods that have been detailed.

Superior frontal convolution.

Ascending frontal convolution.

Ascending parietal convolution.

Angular convolution.

Middle temporo-sphenoidal convolution.

Superior occipital convolution.

Cuneate lobule.

It may be said at the outset that no one of these regions was entirely free from some defect, though this was only slight in the case of the superior frontal and middle temporo-sphenoidal convolutions. In the other convolutions examined the changes were very marked, but those in the ascending parietal appeared to be the most pronounced, although the changes met with in the ascending frontal convolution suggested that they were more advanced than in the other convolutions.

The essential change was one of degeneration of the pyramidal cells of the cortex. These cells were to be seen in all stages of degeneration from comparatively slight, in which with neuraxons and dendrons considerably altered the cytoneurons¹ themselves still preserved some of their original shape (see Figs. 4—7), to a stage so pronounced that, as seen in specimens prepared by the silver method, a black irregular mass is the sole representative of what was once a cell, the positions of such black masses in relation to other cytoneurons less altered being often the only reliable evidence that the masses in question represent cells at all.

Between these extremes all degrees of intermediate changes were met with, some cytoneurons being swollen and globose, others partly broken down, but still retaining some evidence of their original shapes, and so on. Groups of degenerated cytoneurons were often met with, and in such groups were often seen side by side cytoneurons in all

¹ In this paper the term "neuron" is used to denote the cell-body with its different processes, "cytoneuron" denotes the cell-body, "neuraxon" the axis-cylinder process, and "dendrons" all other processes of the cell.

stages of degeneration from very slight to a degree amounting to almost complete extinction of the cell.

The changes in the neuraxons and dendrons were most pronounced. Even in those cases where the cytoneurons, though evidently degenerating, still preserved their pyramidal shape, the dendrons were, as a rule, broken off, as were the neuraxons; or the latter, if still continuous with the cytoneurons, showed the presence of the degenerative process most clearly. The beaded appearance due to breaking up of the axis cylinder was most characteristically seen in some instances (see Fig. 5).

Specimens prepared by the Marchi method showed most extensive degeneration of the fibres of the corona radiata, chiefly in connection with the central convolutions. But that all the fibres had not met with this fate was evident on examination of sections prepared by the Palm method, which revealed the existence of a large proportion of healthy nerve-fibres, even in connection with the ascending parietal convolution, where the degenerative processes in the cells were so pronounced.

With so much degeneration present, it will be readily understood that a large amount of free fatty material was distributed throughout the sections. This fatty debris was also taken up by the perivascular lymphatics, some of which were full of this, as is shown in Figs. 8 and 9.

The changes that have been described relate chiefly to the convolutions other than the superior frontal and middle temporo-sphenoidal, in which, as has been explained, the degenerative changes were only slight. Such changes as were met with in these convolutions were of the same nature, but much less marked, isolated degenerated cells being found (see Fig. 7) rather than any clusters of them, as was the rule in connection with the central and other convolutions examined.

THE CEREBELLUM.

Unlike the cerebrum, the cerebellum presented no evidence of degenerative or other change in the cells of

Purkinje or those of the corpus dentatum. Indeed, no structural change could be detected in connection with the organ, apart from certain degenerated fibres met with in its superior peduncles. As only a small piece of the cerebellum was available for examination, there was no possibility of judging of the exact course or distribution of these degenerated fibres in the organ.

THE PONS.

The pyramids.—The most striking feature presented by sections stained according to the Pal method was degeneration of the pyramidal fibres (see Figs. 10—13). The appearances presented by such sections, as seen by the unaided eye, suggested complete sclerosis of these tracts of fibres; but on microscopical examination many undegenerated nerve-fibres were seen in these areas of sclerosis. Further, specimens prepared by the Marchi method revealed many nerve-fibres in these areas in a recent state of degeneration, such fibres being stained an intense black by the osmic acid.

The fillet.—This structure also presented evidence of degeneration on both sides; the sclerosis as seen in specimens stained by the Pal method was chiefly obvious in the lateral fillet, while the fibres in a state of recent degeneration, as shown by the Marchi method, were much more numerous in the mesial fillet (see Fig. 14). But in neither the lateral nor the mesial fillet was the extent of degeneration in any way comparable to that seen in the case of the pyramidal fibres.

The descending root of the fifth nerve.—Sections prepared by the Marchi method showed unquestionable evidence of recent degeneration in these tracts (see Fig. 15).

Superior cerebellar peduncles.—No obvious change could be detected in these structures as seen in sections prepared by the Pal method; but in specimens prepared by the Marchi method slight scattered degeneration was evident on both sides. The amount of degeneration was, however, slight, and in no way comparable to that seen in the

descending root of the fifth or in the fillet, which, as has already been said, was in its turn much less in amount than that in the pyramidal tracts.

The posterior longitudinal bundles.—These tracts were practically free from degeneration. The bulk of the nerve-fibres were certainly normal, but here and there a few doubtful spots were seen in specimens prepared by the Marchi method, the exact nature of which it was impossible to be certain about. As was first pointed out by Mott,¹ the only way to be certain as to whether such doubtful spots represent degenerated nerve-fibres or not, is to examine them in longitudinal section, a method which, for obvious reasons, could not be adopted in this case.

THE MEDULLA OBLONGATA.

The pyramids.—As in the pons so here the most obvious defect was the appearance of the pyramids as seen by the unaided eye in sections prepared by the Pal method. So complete did the sclerosis appear (see Fig. 16) that it would not have been surprising if not a single healthy fibre had been found on microscopical examination. This was not the actual case, however, as many undegenerated fibres were present in both pyramids. Specimens prepared by the Marchi method revealed numerous fibres in a recent state of degeneration also.

The fillet.—The degeneration of the fillet was best seen at its decussation in specimens stained by the Marchi method. The degenerated fibres cut longitudinally could be traced with great exactitude to the nuclei of the posterior columns, in which they all appeared to terminate. Fig. 17 shows these degenerated fibres in their course at the decussation of the fillet. The degeneration was most symmetrical.

THE SPINAL CORD.

Pyramidal tracts.—Sections prepared by Pal's method showed what appeared to be complete sclerosis of the direct and crossed pyramidal tracts on both sides, as seen

¹ Mott, 'Brain,' 1895, part i, p. 1.

with the unaided eye, and as is shown in Fig. 18, which is a photomicrograph of such a section taken from the cervical region of the spinal cord. On examination under the microscope, however, as in the pyramids at higher levels, so here the pyramidal tracts, both direct and crossed, contained a considerable number of normal nerve-fibres scattered throughout these areas. So, too, in specimens prepared by the Marchi method, a fair number of nerve-fibres in a recent state of degeneration were to be seen. The changes in these tracts, as has just been described, was the same throughout their entire course in the spinal cord.

THE PERIPHERAL NERVES.

The sciatic nerve.—The only peripheral nerve from which a piece was obtained for examination was the sciatic. Most careful microscopical examination of sections of this nerve stained by various methods failed to detect any abnormality in its structure. All the nerve-fibres were apparently absolutely intact, and there was no evidence of interstitial change.

Ocular changes.—We are indebted to Mr. Treacher Collins for his kindness in making a microscopic examination of both eyes. The following is his report.

“The optic nerve of one eye was cut longitudinally and of the other transversely. One description of the conditions found will apply equally to the two eyes.

“The optic nerve is much atrophied; there is considerable increase in the amount of fibrous tissue between the bundles of nerve-fibres, and also a large increase in the number of round-cells in the nerve. In one section a considerable collection of round-cells is seen situated between the central vein and artery at the intra-ocular end of the nerve. There is considerable cupping of the optic disc, due to atrophy of the nerve-fibres almost down to the lamina cribrosa, and not to depression backward of that structure. No inflammatory exudation is seen between the dural and pial sheaths of the nerve. The central

artery is full of blood-clot, the central vein is empty ; no alteration is seen in the walls of either vessel.

"The choroid in the region of the yellow spot has its vessels dilated, but no inflammatory or other changes are seen in it.

"The retina at the yellow spot has a fold or ruck in it (see Fig. 19), so that it is slightly detached in that region from the choroid. It is there much thickened, due to enlargement of the outer molecular layer, the tissue of which is much spaced out (see Fig. 20), here and there cavities being left ; the condition is apparently due to oedema. So far as can be made out, the other layers show no changes. Elsewhere than in the yellow spot the retina appears healthy.

Treatment.—On admission the child was given Potas. Iod. gr. $\frac{1}{2}$ three times a day, which quantity was soon increased to gr. 1 thrice daily, and continued for one month with no apparent ill effect, but without benefit.

On February 1st, 1894, tabloids of cerebrine were administered ; at first 1 grain three times daily, afterwards increased to 10 grains in the twenty-four hours, and continued for two months. All active treatment was then discontinued until the end of June, when in consequence of Andriezen's observations¹ on the nature of the pituitary gland, a fresh sheep's gland was obtained daily, finely minced and pounded, and administered each day for six weeks. No further treatment was carried out beyond such general directions as to food and warmth as the wasted condition of the child demanded. None of the measures adopted appeared to have the slightest effect in arresting the gradual progress of the disease.

CASE 3.—David R—, brother of the preceding patients, born November, 1894. Was seen for the first and only time on February 20th, 1896, being then fifteen months old.

The mother stated that he was older than the other children before any signs of the disease appeared ; in fact,

¹ 'Brit. Med. Journ.,' 1894, vol. i, p. 54.

not until the sixth month was any change noticed, the first sign being the weakness of the back and difficulty in holding his head erect; the enfeeblement subsequently extended to the muscles of the extremities.

When examined, the child lay helpless in bed with its head thrown back, quite unable to move. The forearms were extended, and the hands pronated. If flexed, the arms returned to the extended position.

The legs were extended on the thighs, but the rigidity could be overcome without much force.

The body was wasted. General intelligence was better retained than in the case of the other children, and the child recognised the voices of his parents; but the expression of the face indicated mental enfeeblement, and he frequently laughed without cause.

Hearing was acute; any sudden sound made the child jump violently.

Vision was nearly abolished. Symmetrical changes at the macula similar to those occurring in the other patients were present. There was also distinct optic atrophy.

Dr. F. J. Smith's case.—We are greatly indebted to Dr. Smith for his kindness in allowing us to include in this paper an account of a case of the disease which was under his observation at the London Hospital, and which has not been previously published.

Garvil L—, aged 12 months, the child of healthy parents, both of whom were Jews. There were four other children in the family, all of whom were healthy, and the mother had had no miscarriages.

Nothing abnormal was noticed until the child was three months old, when the parents observed that it was backward as regards mental and bodily development. The child was then taken to the Evelina Hospital, and from there it was referred to Moorfields, where the parents were advised to take it to a general hospital. It was admitted into the London Hospital on February 17th, 1894, when its condition was as follows:—It cried vigorously when disturbed,

moved its limbs freely when attempts were made to examine the fundi; the movements of the arms and legs seemed perfect in execution, though the child did not appear to move them voluntarily except to put its fingers in its mouth. It was quite unable to sit up, and the muscles of the arms and legs were flabby. There was inclination to talipes varus of the left foot.

The superficial plantar reflexes were not brisk, but definite; both knee-jerks were glib.

The fontanelles were closed; the forehead was not particularly square or prominent. The lower median incisor teeth were just protruding through the gums. There was no particular enlargement of the wrists, and the tibiæ, though bowed outwards, especially the right, presented no enlargement of the epiphyses. The rib cartilages were not enlarged.

There was no enlargement of the liver or spleen, nor was there any enlargement of the glands in the neck or groin.

Ocular changes were present, and consisted in a whitish-grey appearance at each yellow-spot region, in the centre of which area was a red spot. The fundus was otherwise normal.

The child gradually wasted, and became progressively weaker. A week after admission difficulty of swallowing became apparent, and steadily increased. No other change was noted up to the time of death, which took place on April 11th, 1894, nearly two months after admission to the hospital.

No special treatment was adopted. A post-mortem examination was not allowed.

CHARACTERISTIC FEATURES OF THE DISEASE.

Etiology.—The disease occurs in both male and female children, and of twenty-three cases recorded, including those which form the subject of this communication, eight have been in boys, eleven in girls, and in four the sex is not stated. No distinct exciting cause can be assigned.

It has no apparent relation to syphilis, consanguinity of marriage, or any of the usual hereditary diatheses.

Racial peculiarity appears to have some influence; in the published instances of the disease in which the nationality has been stated, including all the cases occurring in this country, the children have been Jews.

More than one child of the same parents is usually attacked, but no regularity is observed in the order in which this takes place. It may be the earlier or later children, or every other child, while the other members of the family are quite healthy.

Symptoms and progress.—For convenience of description the symptoms and progress of the disease may be considered as they occur in three stages.

The first stage.—An infant, the subject of this disease, is born at the full term of gestation, and may be well formed and developed, differing in no outward respect from a healthy child, until about the completion of the third month. At this time some weakness of the muscles of the back and neck is observed, and often a suspicion that the child sees imperfectly is entertained. Should the eyes be examined with the ophthalmoscope about the fourth or fifth month, definite and characteristic changes will be discovered in the region of the macula lutea. These will be described more in detail later.

The second stage.—The child is now unable to sit up, its head falls backwards if unsupported; when lying on its back it is unable to turn on to either side. Objects placed in its hands are grasped but feebly, and soon dropped. It generally is apathetic, taking no notice of surrounding objects, and the face bears an expression of mental enfeeblement. Vision is reduced to perception of light, but the sense of hearing is acute, and remains so during life, any sudden sound causing the child to start. The sense of taste is also preserved.

The third stage.—Atrophy of the enfeebled muscles ensues, and soon those of the whole body are involved. Emaciation progresses and becomes most marked. The

deep reflexes are exaggerated, and still later in the course of the disease, rigidity of the extremities and retraction of the head become prominent features; occasional spasmodic contractions cause the child to start and cry from pain. Convulsions have been noted in one or two instances during the course of the disease, but they would appear to be an accidental accompaniment, and are at all events not the rule.

The temperature remains normal throughout the course of the disease. The heart, lungs, and abdominal viscera are also normal.

The ocular symptoms.—The ocular symptoms, which are an early and we believe an absolutely diagnostic sign of the disease, consist in symmetrical changes at the macula lutea, in which situation, covering a space nearly twice the size of the optic disc, is a whitish-grey patch, only slightly raised above the general surface of the retina, somewhat oval in shape (the axis being horizontal) with softened edges. A few retinal vessels are visible on it at the periphery. In the centre of the patch is seen the fovea centralis as a dark cherry-red spot (see Fig. 21). There is no other opacity of the fundus, and one is forcibly reminded of the appearances seen in embolism of the central artery of the retina. In the early months the optic papilla shows no decided changes, and the child can perceive light. Later on there is definite optic atrophy (see Fig. 21), and total amaurosis. It is to be noted, however, that the changes at the macula from the date of their appearance until the close of life remain unaltered. That they are not congenital is proved by an observation of Mr. Tay's ('Trans. Ophthal. Soc.,' vol. iv), and also by the condition found in our second case.

The duration of life.—The duration of life varies from one and a half to two and a half years, but is usually less than two years. All the subjects of this disease are known to have died except two, and they were becoming worse when last seen. It can be well understood that the duration of life must largely depend on the care and attention

bestowed upon the child in checking any intercurrent disorder; but apart from these causes, death is sometimes unexpectedly sudden.

MORBID ANATOMY.

Central nervous system.—Degeneration of the neurons of the cerebral cortex is the fundamental change in these cases. Further, all the evidence points to this change being a primary one of the nerve elements, and in no way secondary to any inflammatory or other process. The appearances met with also indicate a progressive change; even in those parts where it is most advanced there is abundant evidence of the progressive character of the disease as shown by the different degrees of degeneration of the pyramidal cells met with in the same section.

With such changes in the cortex it is not surprising that there should be degeneration of the fibres of the corona radiata, and of the pyramidal tracts throughout their whole course through the pons, medulla, and spinal cord.

The other tracts which we found degenerated were the fillet, the descending root of the fifth nerve to a less degree, and the superior cerebellar peduncles still less; all these structures being affected symmetrically on the two sides.

Eyes.—The retina at the yellow spot is much thickened, this being due to enlargement of the outer molecular layer, the tissue of which is spaced out, the most marked changes occurring in the neighbourhood of the fovea, and becoming less evident towards the periphery of the affected area. The other layers show no changes, and the retina in parts other than the yellow-spot region is healthy.

It seems highly probable that much of the prominence of the macula region of our second case, as seen in the microscopic section, is the result of accidental circumstances connected with the process of preparation of the tissue for microscopic examination. This is suggested by the appearance, by the existence of a similar though much smaller fold of the retina outside the macula area (see Fig. 19), and by the fact that such an obvious elevation could hardly be

overlooked in an ophthalmoscopic examination, whereas neither by ourselves nor by others has any marked swelling of the area been noted during life. There is, however, apart from any accidental circumstance, an increase in the width of the outer molecular layer in the region of the macula.

The optic nerve is atrophied with increase of interstitial connective tissue, and large increase in the number of round-cells in the nerve. There is considerable cupping of the optic disc, the result of atrophy of the nerve-fibres. There is no inflammatory exudation between the dural and pial sheaths of the nerve.

PATHOLOGY.

Central nervous system.—Apart from the clinical evidence that the disease is not congenital, but develops after about the third month of extra-uterine life, the changes met with in the central nervous system preclude the possibility of any such view being entertained. In all the parts of the central nervous system examined in our cases there was nowhere the slightest indication of any congenital defect. All the structures appeared to have been normally developed, and the degenerative changes met with were certainly occurring in normally developed structures. As has been already said, those pyramidal cells in the cerebral cortex in which the changes were least marked appeared of almost normal shapes and sizes, and their arrangement was regular and in every way compatible with a normal state of things prior to the onset of the degeneration in them.

The condition of the pyramidal tract as seen by the unaided eye in specimens prepared by the Pal method strongly suggested a complete absence of any myelinated fibres in these tracts, so that it at once seemed likely that the cortical changes reached an advanced stage early enough to preclude the possibility of the pyramidal fibres becoming myelinated at all. That this view could not be wholly correct was, however, quickly evident on submitting the specimens to microscopical examination, for it then became

clear that many myelinated fibres existed in the pyramidal tracts. Further, the fact that specimens prepared by the Marchi method showed recent degeneration, a method whose reaction depends on the disintegration of the myelin, is additional proof that this view cannot be maintained in its entirety. At the same time it is impossible to deny that some of the pyramidal cells of the cortex may have undergone degeneration early enough to exclude some of the pyramidal fibres from ever becoming myelinated.

The occurrence of changes in the pyramidal tracts in association with the degenerative changes of the pyramidal cells of the cortex cerebri leave very little doubt that the former is dependent on the latter, though the advanced state of degeneration of the pyramidal tracts suggests the possibility that these changes may have preceded those met with in the cerebral cortex. There are, however, no grounds for assuming that the morbid condition is primarily one of the pyramidal tracts which later involves the cortex, if we examine the evidence supplied by the clinical history of the disease, for the cerebral symptoms preceded all those that could be ascribed to sclerosis of the pyramidal tracts.

The degeneration of the direct pyramidal tracts corresponds so exactly in every particular with that met with in the crossed pyramidal tracts that these cases afford important proof that Boyce's observation,¹ that in the cat all the pyramidal fibres decussate and leave no direct pyramidal tract, is not true of man.

In the absence of any detectable lesion of the posterior column nuclei, it must be granted that the change met with in the fillet is a descending degeneration.² But in spite of the marked changes in the cerebral cortex the case cannot be claimed as one showing that fibres pass downward from the cerebral cortex in the fillet directly to the posterior column nuclei, as the basal ganglia were not obtained for examination. All that the case proves in this

¹ Boyce, 'Phil. Trans. Roy. Soc.,' vol. 186, 1895, B, p. 321.

² Cf. Monakow, 'Neurol. Centralblatt,' 1885, p. 65; and Flechsig and Hösel, 'Archiv f. Psychiatrie,' Bd. xxiv, Heft 8.

respect, then, is that fibres do certainly appear to degenerate downward or caudalward in the fillet, but as to whether such fibres come directly from the cerebral cortex or whether from the basal ganglia must be left an open question.

The same must be said of the degeneration met with in the descending root of the fifth nerve, and, in the absence of any primary changes in the cerebellum, of the degenerated fibres seen in the superior cerebellar peduncles.

Eyes.—The precise relationship of the ocular changes to those met with in the central nervous system is not very evident. The statement, however, applies more to the changes at the macula than to the atrophy of the optic nerve, for this latter condition is commonly associated with certain degenerative diseases of the central nervous system. In spite of Mr. Waren Tay's experience in the early stages of one case, the atrophy must, we think, be looked on as a primary one and not secondary to neuritis; it certainly was so in our own cases, and it has all the characters of a primary rather than of a post-neuritic atrophy. With regard to the changes at the macula, it is difficult to understand why the outer molecular layer should especially be affected. It is just possible that the changes are due primarily to a degeneration of the ganglion cells of the retina similar to that met with in the pyramidal cells of the cerebral cortex, and that the limited ophthalmoscopic appearance is partly due to the much greater abundance of these cells in the macular region; but of this we have no evidence, as unfortunately the specimens were not prepared by methods specially calculated to reveal the existence of such changes. One thing is, however, quite certain, and that is that the changes at the macula are not of embolic origin.

Doubtful as is the nature of the relationship between the ocular changes and those of the central nervous system, the relationship of the ocular changes to each other is no less obscure: whether the optic atrophy is secondary to and dependent on the changes at the macula; or whether, as seems more probable to us, they are both dependent on a common cause related to the changes met with in

the cerebral cortex, must, for the present, be left undecided.

Treatment.—In view of our imperfect knowledge of the primary cause of this disease, any treatment adopted must be more or less experimental. We, at any rate, now know the degenerative changes which are in progress, and must endeavour to combat them. We also know that if one member of the family has been attacked, some other children of the same family will almost certainly suffer. Can we therefore prevent its occurrence by the administration of any remedies to the mother when *enceinte*? The only attempt which we have made in this direction was by giving the mother Potass. Iodidi gr. v thrice daily during the whole period of two pregnancies; but the result was negative, as one child was healthy but the other has been attacked.

For the same reason it would seem advisable that where the one child is known to have suffered, subsequent infants should be weaned from the time of birth. We are not aware whether this has been carried out; our own patients were nursed by their mother.

ADDENDUM.

When our paper was presented to the Society in March, 1896, the fifth child of the family, though affected, was still alive, and the sixth child when examined in February, 1896, appeared healthy. The sixth child has since become affected, while the fifth child died on May 6th, 1896, and a microscopical examination of the central nervous system has been made. We have, therefore, altered the account of the history of the family, which is to be found at the commencement of the paper, in accordance with the state of the family now as opposed to the condition of things which existed last March.

We now propose to add the briefest possible clinical report of the terminal stages of the illness of the fifth child (Case 3 in the paper), to note the date when the

disease was detected in the sixth child of the family, and to give a short account of the autopsy and subsequent microscopical examination of the nervous system in Case 3.

David R— was first seen when three months old, at which time he presented no evidence of the disease. The patient was not seen again until March 8th, 1896, when he was admitted into the Children's Hospital at Nottingham, being then fifteen months old. The symptoms, both general and ocular, were similar to those already described, but from the mother's statement they would appear to have been later in commencing, viz. at the sixth month.

The child was fairly nourished, and weighed 14½ lbs. Paresis of the limbs existed, and was associated with occasional rigidity, but there was no permanent flexion or extension consequent on muscular spasm.

The patient remained in much the same condition until four days before death, when he gradually became unconscious, and died on May 6th, 1896. There were no convulsions, nor was there any rise of temperature.

Autopsy.—An examination of the head was made five and a half hours after death. There was no thickening, opacity, or adhesions of any of the cerebral membranes. The brain weighed 2 lbs. 2 oz., was firm in consistence, and the sulci were compressed. There was no excess of cerebro-spinal fluid. Portions of different regions of the cerebral cortex, the basal ganglia, pons, medulla, upper part of spinal cord, and cerebellum, together with the posterior halves of the eyes, were at once placed in Müller's fluid for preservation and subsequent microscopical examination.

Microscopical Examination.

Cortex cerebri.—Extensive degeneration of the pyramidal cells of the cortex was found, but the condition was evidently less advanced than in Case 2, as there were no evidences of fatty disintegration of the cells, and the perivascular lymphatics did not contain fatty débris, as

was the case in the other patient's brain. Although not so advanced, the changes were none the less pronounced, as was best shown by specimens prepared by Nissl's method. The pyramidal cells were variously altered in shape, the majority being so distended as to have entirely lost their pyramidal shape, and to have become balloon-shaped (see Fig. 22); others, while distended, preserved some slight traces of their former pyramidal shape; while some cytoneurons showed evidences of commencing disintegration at parts. The granules of the cells were in some instances collected around the nucleus, while in the less altered cytoneurons fine granules could still be seen scattered in their interior, but all coarse granules had disappeared. Some of the cells appeared to have entirely lost their granules, a process of vacuolation of the cytoneuron having taken place; and in others the granules appeared to have run together more or less at the periphery of the cell. Many phagocytes lying in close relationship to the pyramidal cells contained deeply stained granules, while others of these phagocytes were to be seen round the vessels of the cortex. The nerve-fibres were degenerated, and the general appearance presented by the meshwork of interlacing fibres suggested an abnormal spacing out and separation of the various elements of the cortex, such as might reasonably be supposed to have been brought about by an œdematous condition of these parts.

Basal ganglia.—Nothing abnormal could be detected in connection with the basal ganglia, but the fibres of the internal capsule were degenerated.

Pons.—Well-marked degeneration of the pyramidal fibres was traced through the pons, both as shown by a modification of Pal's method, and as seen in specimens prepared by the Marchi method. Degeneration of the fillet could also be traced through the pons.

Medulla oblongata.—The degeneration of the pyramids was well seen throughout the medulla, and the degeneration of the fillet was strikingly evident at the decussation of this structure, its recently degenerated fibres, stained

by the Marchi method, being seen in longitudinal section, passing between the interolivary layer and the posterior column nuclei. The cells of the posterior column nuclei showed no evidence of degeneration.

Spinal cord.—The portion of the cervical region of the spinal cord which was available for examination showed well-marked degeneration of both crossed and direct pyramidal tracts. All other tracts of the spinal cord in this region were intact, and no alteration in the grey matter could be detected.

Optic nerves.—There was well-marked atrophy of the optic nerves.

R—, a boy aged 8 months, another member of the same family, was kindly examined for us by Mr. Treacher Collins and Mr. Tay, at Moorfields Hospital, on July 23rd, 1896, when the characteristic changes were found at each macula.¹

Remarks.—It is interesting to find that the changes met with in the central nervous system of Case 3 so closely correspond to those that were met with in connection with Case 2. Of still greater interest, however, is the fact that Case 3 died in an earlier stage of the disease than did Case 2, and that the changes met with on microscopical examination of the cortex cerebri in Case 3 were correspondingly representative of an earlier stage in the process of cerebral degeneration than those met with in connection with Case 2.

The possibility that many of the pyramidal fibres may never have become myelinated is still more forcibly suggested to us by the last case examined than by the former one, in that with more recent changes in the cerebral cortex, the apparent sclerosis of the pyramidal tracts in the pons, medulla, and spinal cord is extensive.

¹ On January 12th, 1897, there was some inability to hold up the head, and to sit up unsupported. Objects were grasped feebly. The limb muscles were flabby, but responded normally to the faradic and galvanic currents. There was moderate flexor spasm of the hands and feet. The knee-jerks were present, but not exaggerated. Cutaneous sensibility appeared to be preserved.

In that, so far as has been discovered, the lesion is a purely cortical one, the degeneration of the fillet is of interest, the evidence supplied by Cases 2 and 3 supporting the view that a cortical fillet exists, and that it is capable of degenerating downwards. Certain it is that in neither case could any lesion of the posterior column nuclei be discovered, and that though in Case 2 the basal ganglia were not examined, an examination of these structures in Case 3 showed no change.

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(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 37.)

DESCRIPTION OF PLATE XVI.

Infantile Cerebral Degeneration with Symmetrical Changes at the Macula (E. C. KINGDON and J. S. RISIEN RUSSELL).

FIG. 4.—A photomicrograph showing degenerated pyramidal cells from the ascending frontal convolution. Most of the cytoneurons¹ have been destroyed to a degree past recognition, but there are three on the right side of the figure which have preserved some of the characters by which they can be recognised as cells, though that farthest to the right is almost beyond this stage.

FIG. 5.—In this figure also, three cytoneurons can still be recognised as such—two towards the middle line in the lower half of the photograph, and the third to the left of these and at a higher level. The latter shows the broken-up, beaded appearance of its neuraxon, a condition far better seen under the microscope than it has been possible to reproduce in the photomicrograph, which was taken from a specimen of the ascending parietal convolution.

FIG. 6.—Only one cytoneuron near the centre of the photograph has escaped alteration beyond recognition; its dendrons are broken off short, and but a slender remnant of its neuraxon is still to be seen. There are other possible cells in this figure, but they have reached a stage of disintegration which makes it impossible for a positive opinion to be expressed about them.

FIG. 7.—This is an example of an isolated degenerated cytoneuron. Its dendrons are broken off short, but a considerable length of the neuraxon is still connected with the degenerated cytoneuron.

FIG. 8.—This photomicrograph shows a blood-vessel in the middle of the field with its perivascular lymphatic space full of fatty débris stained black by osmic acid.

FIG. 9.—This figure represents another vessel seen under a higher power of the microscope than the last. The outline of the vessel can be well seen, with the globules of fatty débris in its perivascular lymphatic space.

¹ "Neuron" = the cell-body with its different processes; "cytoneuron" = the cell-body; "neuraxon" = the axis-cylinder process; and "dendrons" = all the other processes of the cell.



FIG. 4.



FIG. 5.



FIG. 6.

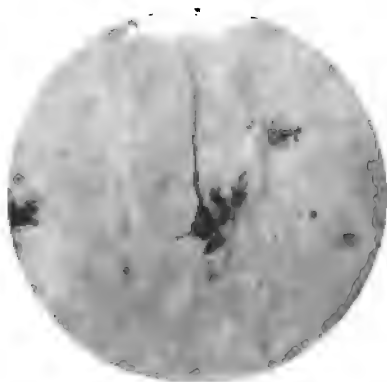


FIG. 7.

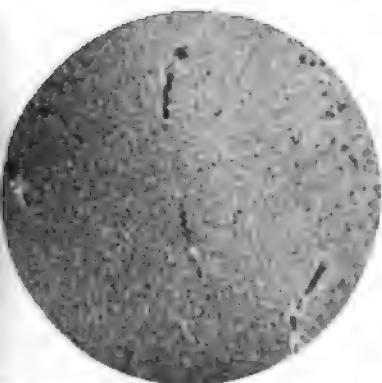


FIG. 8.



FIG. 9.

DESCRIPTION OF PLATE XVII.

Infantile Cerebral Degeneration with Symmetrical Changes at the Macula (E. C. KINGDON and J. S. RISIEN RUSSELL).

FIGS. 10, 11, 12, and 13.—In all these photographs, which have been taken from specimens stained by the Pal method, the sclerosis of the pyramidal fibres is evident; and in Figs. 10 and 11 some sclerosis of the lateral fillet can also be made out.

FIG. 16.—What appears to be complete sclerosis of the pyramids in the medulla oblongata is shown in this photograph of a specimen stained by the Pal method.

FIG. 18.—The sclerosis of both direct and crossed pyramidal tracts, as seen in transverse section in the cervical region of the spinal cord, is well shown in the photograph, also taken from a specimen stained by the Pal method.



FIG. 10.

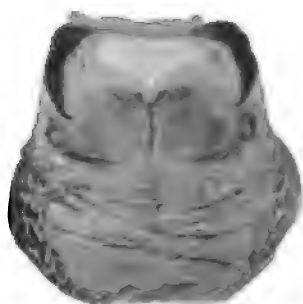


FIG. 11.



FIG. 12.

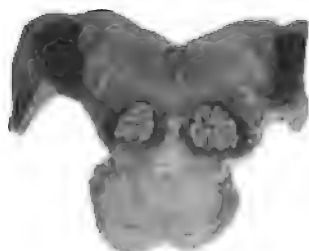


FIG. 13.

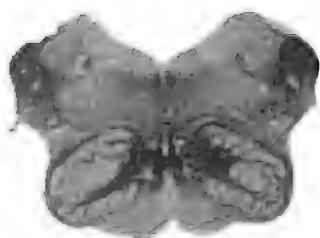
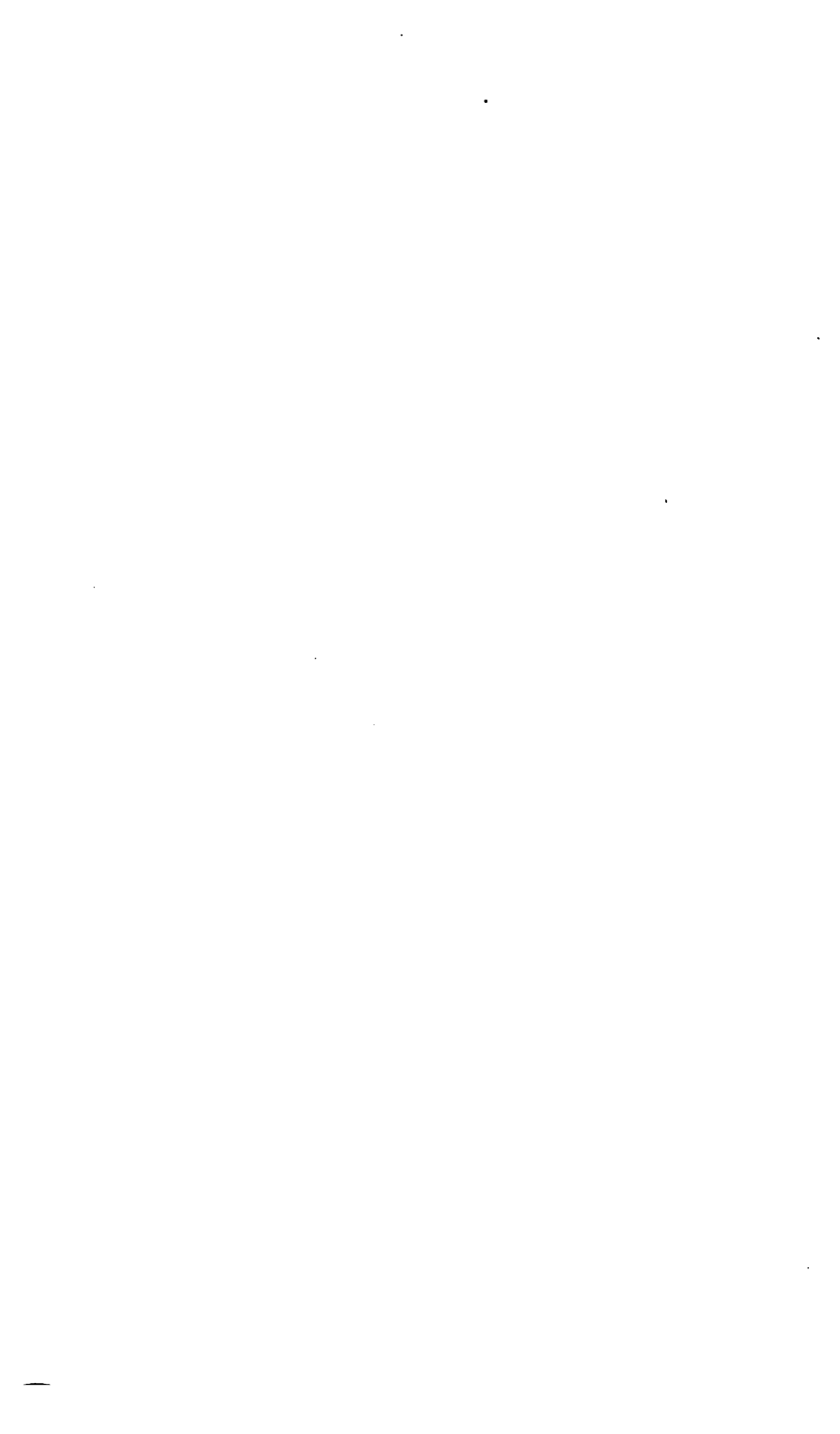


FIG. 16.



FIG. 18.





DESCRIPTION OF PLATE XVIII.

Infantile Cerebral Degeneration with Symmetrical Changes at the Macula (E. C. KINGDON and J. S. RISIEN RUSSELL).

FIG. 14.—A portion of the mesial fillet is seen in this photomicrograph, with scattered degenerated fibres stained black by the Marchi method, and seen as black dots on transverse section.

FIG. 15.—The black dots in the centre of this figure represent degenerated fibres in the descending root of the fifth nerve as seen on transverse section in a specimen stained by the Marchi method.

FIG 17.—The degenerated fibres of the fillet are well seen, many of them in longitudinal section at the decussation of the fillet. The opposite course of the fibres from the two sides as they cross in the middle line can be readily made out.

FIG. 22.—The altered characters of the cytoneurons of the cortex of the ascending parietal convolution in Case 3.



FIG. 14.



FIG. 15.

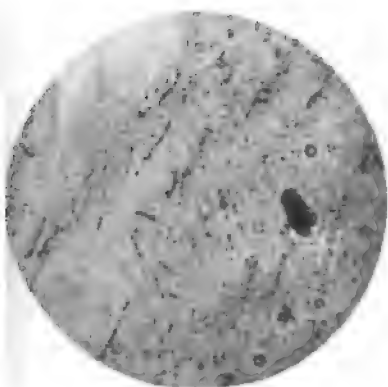


FIG. 17.

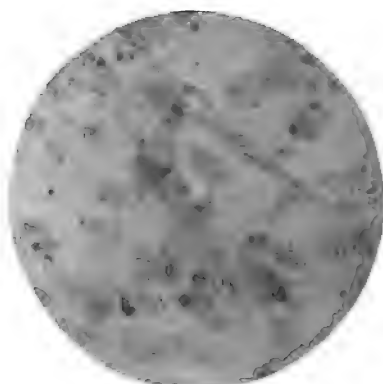


FIG. 22.





DESCRIPTION OF PLATE XIX.

Infantile Cerebral Degeneration with Symmetrical Changes at the Macula (E. C. KINGDON and J. S. RISIEN RUSSELL).

FIG. 19.—The fold in the macula region which was met with on microscopical examination in Case 2 is shown in this photomicrograph.

FIG. 20.—A portion of the retina in the macula region under a higher power of the microscope than the last; it shows the increase in depth of the outer molecular layer.

FIG. 21.—The characteristic appearances in the macular region, as seen with the ophthalmoscope during life, are indicated in this figure, in which the optic atrophy is also represented.

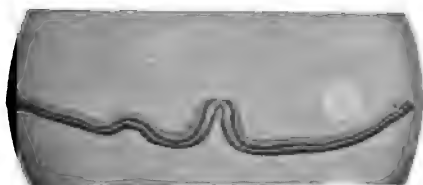


FIG. 19.



FIG. 20.

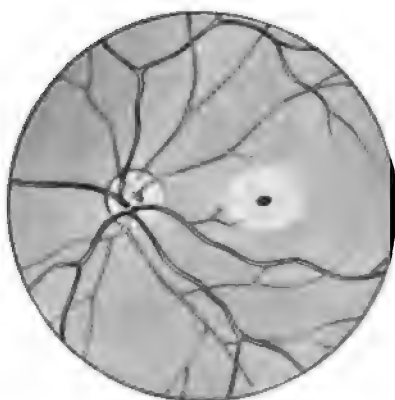


FIG. 21.



A CASE
OF
PERFORATING TYPHOID ULCER
TREATED BY
OPERATION AND SUTURE, AND RESULTING IN
RECOVERY
BY
THOMAS LAUDER BRUNTON, M.D., F.R.S.
AND
ANTHONY BOWLBY, F.R.C.S.

Received May 4th, 1896—Read January 26th, 1897

JOHN DUGGIN, aged 37, was admitted into Rahere Ward, St. Bartholomew's Hospital, under Dr. Lauder Brunton, on October 3rd, 1895. He was then suffering from diarrhœa. In the beginning of September he had a cough and pain in the chest. On September 20th these symptoms got worse, and the patient had a headache. There was no shivering or vomiting. On the 26th was obliged to go to bed. On this day he vomited. The coughing continued. On September 28th the bowels were open two or three times a day, and diarrhœa continued till admission.

On admission he seemed semi-comatose, with a tempe-
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perature of 103.4° , pale face, and dry, brown, cracked tongue. The abdomen was moderately distended. The liver and spleen were not felt, but were apparently very tender. The bowels were open four times. The motions were light yellow, free from blood. There was gurgling in the right iliac fossa. The respirations were 28; loose cough; no expectoration; no dulness but rhonchus over the whole chest. The cardiac sounds were apparently free from any murmur. The case was diagnosed as one of typhoid fever. On the day after admission he passed two or three ounces of bright blood. The bronchitis continued, and on the 15th there was some bronchial breathing at the right base, which appeared to be due to collapse. The temperature was very irregular. On October 24th it was nearly normal, and continued so until the 30th, when it again rose. Typhoid spots came out and the spleen was tender. The temperature fell again on November 12th, and remained subnormal until December 7th, when it rose to 99.8° in the evening. On the 8th it was 100.2° . On the 9th it was 99.4° . Each day the temperature fell in the morning nearly to 98° . The next three days it never rose above 99° . The lungs were clear, the spleen could not be felt, although there was some tenderness in the splenic region, and there were no spots.

On the morning of December 14th, at three a.m., the patient woke up complaining of very severe pain in the abdomen. He complained of feeling cold, and his temperature sank to 97° . After the application of a large poultice the pain became easier, and he went to sleep. On awakening he still complained of bad griping pain, and about 8 a.m. the bowels were opened and a natural motion passed with some relief. At 12.45 p.m. he was seen by the house physician. He was then complaining of severe abdominal pain, which was worst in the epigastrium. The temperature was now 102° ; pulse 110, soft. There was slight distension of the abdomen below the umbilicus, but the upper part

moved well on respiration. He was very tender to deep palpation, and on deep inspiration or coughing. The liver dulness was normal. The patient lay on his side with his legs drawn up, but he could lie straight on his back without pain. He had not been sick or complained of nausea, and he had taken liquid food without trouble. At 3 p.m. he was seen by Dr. Brunton, who considered that an operation might be required later on, and asked Mr. Bowlby to see the patient at 5 p.m. There was not then much change in his condition except that he was rather more easy. There was still no sickness. At 8.30 p.m. he was again seen by Dr. Lauder Brunton. The general condition was still good, and there was no sickness, but the distension of the lower part of the abdomen had increased, and abdominal respiration was much more limited. Flatus was retained, and the liver dulness was still natural. It was considered that the symptoms were probably due to perforation of the intestine at the seat of an ulcer, but it was evident that if this was the case there were adhesions limiting the escape of the contents of the bowel to the lower part of the peritoneal cavity. As the symptoms were ingravescent Dr. Brunton decided that laparotomy ought to be done without further delay, and the operation was accordingly performed as soon as possible after this by Mr. Bowlby. Mr. H. Marshall acted as assistant during the operation.

The patient was placed under the influence of chloroform, and the abdomen was opened in the middle line below the umbilicus. The upper part of the peritoneal cavity was free from gas or faecal matter, but the great omentum was adherent in the pelvis, and as soon as it was separated from the subjacent intestine it was found that the whole of the intestines beneath it were matted together. On freeing the uppermost coils about half a pint of fluid was set free. It was dirty yellow in colour, and evidently consisted of some intestinal contents mingled with peritoneal exudation. On passing the hand into the cavity amongst the intestines from which this fluid had

escaped, a very indurated coil of bowel could be felt, and after a little trouble was separated from its adhesions and drawn out of the abdomen. It was then seen that the gut had been perforated by a small ulcer, about as large as a pea, situated opposite to the attachment of the mesentery. This ulcer was evidently in the centre of an inflamed Peyer's patch, for an area of induration corresponding in size to such a patch could be felt in the wall of the bowel. The whole coil of intestine was covered with fibrin and recent lymph, and was thickened and oedematous. This lymph was removed as thoroughly as possible, and the whole bowel very carefully washed. Ten silk sutures were then passed after Lembert's method, transversely to the long axis of the bowel, which was clamped by the fingers of the assistant. The sutures furthest from the ulcer were passed clear of the indurated Peyer's patch, so that the line of suture was about an inch and a half in length. Flatus and fluid matter were then allowed to enter the sutured coil of gut, and it was found that there was no leakage, and that the lumen was free. The abdomen was finally irrigated with hot water until all foreign matter seemed to be quite removed, and a drainage-tube having been passed into the pelvis, the wound was sutured. The operation lasted about forty minutes, and the patient was not at all collapsed at its conclusion.

Recovery was uninterrupted, the temperature remaining normal throughout. The tube was removed in forty-eight hours, and for four days no food was given by the mouth, except water in teaspoonful doses. The bowels acted on the fifth day, after the use of an enema, and for the next month the patient was very carefully dieted. He left the hospital on February 21st in good health, and returned to his work in April. When seen on April 15th he had regained his normal weight, and the bowels acted regularly. For several weeks after operation he had a slight "dragging pain" in the abdomen and a feeling of tightness, but this gradually passed away.

There is but little to be said with regard to the operation itself. It was performed with the precautions which are customary in all cases of abdominal section for peritoneal inflammations, and the only point besides the method of suture which requires comment is the cleansing of the affected intestine from adherent lymph. As has been already mentioned, there was a considerable quantity of this material present, and it was attached more firmly to the bowel than is common in cases of perforation. It was rubbed off with sponges until the peritoneal coat was quite clean, for it was felt that in the meshes of the lymph there must necessarily remain some of the septic material in which it had been soaked, and no amount of mere irrigation could have detached it. It seems probable that the uninterrupted recovery after the operation may be in part due to the complete removal in this way of a possible source of septic inflammation.

With regard to the actual treatment of the perforation and the passage of the sutures, it was found that there were two difficulties to be overcome. The first of these was the friability of the recently inflamed intestine, and the second the induration of the affected Peyer's patch and the consequent obstruction offered to the inversion of the bowel and the approximation of the peritoneal coats. To overcome these difficulties it was found necessary to pass the sutures at some distance from the perforation, and to draw the peritoneum from the lateral aspects of the bowel over both the puncture and the indurated Peyer's patch, for the latter remained like a rigid body in the substance of the intestinal wall, and quite prevented any doubling in of that part where the perforation itself was situated. In consequence, however, of this method of suture the lumen of the bowel at the site of operation was materially diminished, and it was evident that some permanent narrowing would probably result. It did not, however, appear likely that the narrowing would be sufficient to cause serious stricture, and it is to be noticed that now a year after operation the bowels act regularly,

and there is no evidence that the calibre of the intestine is obstructed.

It is nevertheless certain that these difficulties in suturing are liable to be met with again in other and similar cases, and it is quite probable that in some of them this friability of the intestine and the rigidity of the subjacent Peyer's patch may make suture impossible. But, whilst it is evidently premature to come to any decision as to the best method to adopt under these circumstances, it may be pointed out that the peritoneal cavity could be shut off quite safely from the intestinal lumen in such a case by the suture of the bowel around the perforation to the parietal peritoneum, and the consequent formation of a faecal fistula, which might be left to heal or might require a subsequent operation for its closure.

Another alternative might be preferred, namely, the resection of the affected area of bowel, and this resection might be limited to that part of the bowel containing the affected Peyer's patch, or might be extended so as to comprise the whole coil of intestine. It is not likely that either of these latter procedures would be frequently adopted, for the condition of the patient would, as a rule, demand that the operation should not be prolonged, and there seems but little objection to suturing the bowel to the parietes, and leaving the perforation open, for this could be done with but very little loss of time and without material difficulty.

In conclusion we may point out that the case was one unusually well adapted for operative treatment, and cannot be compared with those cases where perforation occurs during the height of the fever, and in a patient greatly prostrated by the disease. It is probable that the aperture in the bowel had really existed for some time, and had been temporarily but insecurely closed by adhesions. These had yielded when a fuller diet was allowed, and had permitted an escape of the contents of the intestine.

The diagnosis of this case was tolerably clear, but the

absence of any vomiting or nausea, the natural action of the bowels some time after the pain came on, and the absence of any evidence of free gas in the peritoneum combined to create some doubt when the patient was first seen. It was evident after a short time that there was commencing peritonitis in the lower part of the abdomen, and although it is true that this may occur as a complication of typhoid apart from perforation, the history of the case rendered the presence of a perforation more than probable. Whether such cases can recover if left alone is a question which has been frequently debated, but it is now generally conceded that recovery after perforation is of extreme rarity, and not to be anticipated in any given case.

We do not propose to criticise in detail the experiences of other practitioners, but it may be pointed out that there appear to have been up to the present three successful cases of operation on perforated typhoid ulcers. A review of all recorded cases by Dr. Robert Abbe appeared in the New York Medical Record for January 5th, 1895, and from this it appears that the successful operators have been Van Hook ('Philadelphia Medical News,' vol. ix, p. 591), Abbe (loc. cit.), and Netschajew ('St. Petersburg med. Woch.,' 1894, No. 36, Supplement No. 8, p. 46). The last surgeon resected the affected portion of bowel and sutured the divided ends.

Two operations have been recorded in England, by Bland Sutton and Allingham, but in each case the patient's condition was evidently very bad, and the results were not satisfactory. It would appear that the case here described is the third recorded case of the kind submitted to operation in this country, and the first to arrive at a satisfactory termination.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 42.)



TYPHOID FEVER

CONVALESCENCE; SYMPTOMS OF PERFORATION;
LAPAROTOMY; NO LESION FOUND; RECOVERY

BY

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AND

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PERFORATION of the intestine in or after typhoid fever is without doubt usually, and in the opinion of most physicians almost invariably, fatal. But a good many cases have now been recorded in which, though the symptoms which indicate perforation have occurred, the patient has recovered. That any free escape of liquid, or even of gas, into the general peritoneal cavity from the intestines is invariably fatal will probably not be disputed, and in the above cases of recovery it has been supposed that the poison has been limited by adhesions to a small part of the peritoneum. The localised plastic peritonitis that one occasionally finds opposite the ulcers of typhoid fever certainly renders this view possible. But the evidence must needs, in patients who recover, be very inconclusive, and in an able and exhaustive paper upon the subject Fitz ('Trans. Assoc. Amer. Phys.,' vi, 200) has argued strongly against it. He points out that

sudden pain, vomiting, collapse, and fall or rise of temperature are but signs of peritonitis, and he maintains that the absence of hepatic dulness, even when it has been known to be present before, and the abdominal murmur which some physicians have noted, are signs of doubtful meaning and of inconstant occurrence. But peritonitis, he says, may be due to other causes; and after enumerating many, he goes on to argue that from the symptoms mentioned it is probable that most of the recorded cases of recovery have been cases of appendicitis, caused by typhoid lesion of the vermiform process. His proof of this, however, is weak, and we need more records of the state of the appendix in typhoid fever than we now possess before this theory can be accepted.

In January of this year a case fell to our lot, which shows that the same group of symptoms may occur not only without perforation, but even without peritonitis.

CASE.—Jane P—, aged 13, was admitted to Mary Ward in St. Bartholomew's Hospital, under Dr. Hensley, on January 2nd, 1896. She was a healthy-looking girl, but had been feeling tired and had had a cough since December 18th, 1895, and had been in bed since December 25th. When admitted she had some bronchitis, the heart was natural and the pulse fair, the abdomen was rather full, the spleen was not palpable, and there were a good many typhoid spots.

The temperature fell very soon after admission, and was normal on January 9th, which was probably the twenty-second day of her illness. The motions were then solid, the tongue clean, and the pulse 80. The abdomen remained a little full.

On January 15th the patient was very well. Her temperature had been subnormal for seven days, she had been allowed bread and milk for the last two days, and had been taking food well. The bowels had been open on January 4th, 6th, and 7th, but not again until January 14th. They had not been open on the 15th. At 5 p.m. on that day she suddenly complained of acute abdominal pain,

which caused her to cry out constantly. The pain gradually became worse. She vomited frequently. Her pulse was 140 and small. She was given $\frac{1}{4}$ grain of morphine subcutaneously, which relieved her for rather more than an hour; but the pain then returned as badly as before. She was given an oil enema, but without effect in opening the bowels. The abdomen was then full and tense; its walls were rigid, and scarcely moved with respiration; the liver dulness was natural. There was marked tenderness on palpation, especially near the umbilicus, and it was to this region that the pain was referred. The temperature had remained stationary. There was a general aspect of considerable collapse. At 8.30 p.m. we saw the case together, one of us being at the time on duty for Dr. Hensley. The symptoms had in no way improved. The child lay with the thighs drawn up, and screamed with pain on the slightest movement. The abdomen was rigid, and so tender that no complete examination could be made. Retching was almost constant, though but little was now actually vomited. The pulse was still rapid, the hands and feet were cold and clammy.

As it was considered that these symptoms indicated perforation, and as the patient had previously been in good condition, it was decided to open the abdomen.

The patient was placed under the influence of chloroform, and the abdomen was opened in the middle line below the umbilicus. There was no gas or fluid in the peritoneal cavity, and an examination of the cæcum and the last two or three feet of the small intestine showed no evidence of inflammation or perforation. The whole of the colon contained scybalous masses. The abdominal wound was sutured, and a soap and water enema was administered before the patient recovered from the anæsthesia. The bowels were freely open at once. When the patient again became sensible she complained of but little pain, and such as there was quickly yielded to small doses of opium. The bowels were difficult to

move for several days, but became more regular under treatment by glycerine enemata and cascara sagrada. Recovery was uneventful and complete.

This was apparently, therefore, a case of mere colic. Yet it gave rise to symptoms like those which accompany peritonitis, sudden and severe enough to lead us to believe this condition to be present. We have no doubt that our diagnosis would have been confirmed by most physicians. If peritonitis existed, then, although other possible causes deserved consideration, yet, under the circumstances, perforation was far the most probable. If perforation existed the chances were very greatly against recovery if the case were left to itself. On the other hand, the patient had been without fever for seven days, so that we believed the gut would not be swollen or rotten, but sound and fit to heal after a wound, and she was in good case and well able to bear the shock of the operation. Agreeing that had she been in the acute stage of the fever we should have thought an operation almost hopeless, we agreed also that in her actual condition it had a good chance of success.

We record the case partly as a singular and, as far as we can find, unique instance of simulated perforation; partly to show that a recent attack of typhoid fever need be no bar to opening the abdomen should circumstances require the operation.

(For report of the discussion on this paper. see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 42.)

ON JAUNDICE

AND ON

PERFORATION OF THE GALL-BLADDER IN TYPHOID FEVER

BY

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CASES of jaundice with typhoid fever, arising either during its course or during convalescence, are not frequently met with. Such a condition is in fact *rare*; hence Sir William Jenner¹ writes, "Jaundice is a *very* rare symptom in typhus and typhoid fever, so rare that I cannot call to mind a single case in which I have observed it." Murchison² says "I have met with jaundice in four cases of enteric fever." Osler³ writes, "Jaundice is a very rare complication; . . . it was present in only 1.1 per cent. of the Munich autopsies." Frerichs⁴ says, "In ileo-typhus jaundice is only observed in exceptional cases." Louis⁵ quotes two cases, Andral⁶ one, and Liebermeister⁷

¹ 'On Fevers and Diphtheria,' *vide* foot-note, p. 353.

² 'Diseases of the Liver,' p. 456.

³ 'Principles and Practice of Medicine.'

⁴ 'Diseases of the Liver' (New Sydenham Society), vol. i, pp. 172 and 215.

⁵ 'Recherches de Fièvre Typhoïde,' 1841, vol. i, pp. 118 and 367.

⁶ 'Clinique Médicale' (trans. by D. Spillan), 1836, p. 613, case 1.

⁷ 'Cyclopædia of Medicine,' by Ziemssen, vol. i, p. 158.

writes, "Icterus occurs less frequently during typhoid fever than in many other febrile affections, as, for instance, pneumonia." In searching through literature I can find but nine¹ cases recorded as illustrative of this complication. From the facts thus obtained I find that as regards *age*, no case has been observed in any patient under eighteen years of age; it may, however, occur at any age above this up to fifty-four. As to *sex*,² males are much more liable to develop this symptom than females, only one case occurring in the latter. *The time of the occurrence*³ of jaundice varies; thus in the case recorded by Andral the countenance was yellow on the "third day." In other cases jaundice was noted respectively on the "fifth day," "the fourteenth," at the "acme of the fever," the "twenty-sixth day," "twenty-ninth," on the "thirty-eighth day," and, in one case, during a relapse.

Associated symptoms.—Delirium, violent in some instances. Vomiting of bile, in one case continuously for five days. Diarrhœa was observed in some cases, but in two the condition of the bowels was described as being "confined." In one case green-coloured evacuations were noted, but in others the stools were pale or clay-coloured. Bile was present in the urine in most instances, and in one, in addition, "albumen." In one case the urine was passed involuntarily. Other symptoms noted were "epistaxis" so profuse as to require plugging of the nostrils, bloody expectoration in one case without any signs of pulmonary complication. Pneumonia was noted in another case, but

¹ Murchison, 3 cases. Jenner, p. 445 ('On Fevers, &c.'), mentions the case of a soldier in a native regiment at Sierra Leone, and adds, "... it is probable that in some countries jaundice does occur in typhoid fever, &c." Frerichs, 2 cases. Louis, 2 cases. Andral, 1 case. Liebermeister quotes the following: "Griesinger reports ten attacks of jaundice in 600 typhoid fever patients." Hoffman, 10 cases in 250 post-mortems. "In the Hospital at Basle, icterus was observed twenty-six times in 1420 cases."

² In the 26 cases in the Basle Hospital, fourteen were males and twelve females.

³ Murchison mentions that in two of his cases the jaundice appeared during the "primary fever;" both cases died.

the jaundice preceded it by several days. Parotid bubo in one case. Thrombosis of femoral vein (left) in one case, and erysipelas of legs in another. It is of interest to note that in no one case is it recorded that hæmorrhage from the bowels occurred.

As to the condition of the liver in these cases. In instances where any mention has been made it has been stated that there was tenderness on palpation over the hepatic region, and physical examination would tend to show diminution of the hepatic area; in one case it is noted that "hepatic dulness disappeared entirely."

Result of the cases and duration of the jaundice.—In two cases recovery followed; in one the jaundice developed during a relapse and continued for seven days; in the second case, to quote Murchison, "the jaundice came on suddenly about the acme of the fever; . . . the deep jaundice persisted for some time, even during convalescence." The other cases all terminated fatally. In the case where jaundice appeared on the third day, it remained persistent for six days, when the patient died from pneumonia. When jaundice occurred on the fifth day, the patient died three days later in a state of coma, the jaundice being persistent. The instance where the jaundice appeared on the fourteenth day is of considerable interest, inasmuch as it was apparently persistent for fifty-nine days and then disappeared, and the patient did not die for some two months later. In the other cases death occurred in one fourteen days after the jaundice was first noticed, and on the fortieth day of the disease. In the other two cases the jaundice appeared four days before and remained till death, the patients dying on the thirty-third and forty-second days respectively.

Causes of the jaundice.—Writers who refer to this symptom mention two factors in its causation: (1) a catarrhal process; (2) parenchymatous changes in the liver. In one case cited by Louis the liver is described as healthy, and the bile-ducts pervious." In one of

Frerichs' cases, while the bile-ducts were pervious and the gall-bladder contained a small quantity of thin pale bile, the liver itself was unusually soft, and on section, rounded masses, one to one and a half inches in their transverse diameter, were observed; they were soft, almost pultaceous, and separated from the surrounding tissue by a sharp line of demarcation. In the second case described by Louis the bile-ducts were normal, but there were "*tumeurs purulentes ou non purulentes dans la foie.*" In two of Murchison's cases the liver was found to be small and its secreting cells loaded with oil. In the other case described by Frerichs, he places it under the illustrative cases of "*Acute atrophy of the liver,*" and in his remarks says, "*It may be a matter of question whether this case . . . is to be regarded as one of acute yellow atrophy of the liver, or as one of severe (abdominal) typhus, complicated with jaundice.*" It might be added that in this case there were white submucous deposits in Peyer's patches and the solitary glands, the more extensive the nearer the ileo-cæcal valve was approached.

In conjunction with these facts, the following case of jaundice arising during the course of typhoid fever is of some considerable interest.

A female aged 18 years, a gipsy, was admitted into the Adelaide Ward, Royal Berkshire Hospital, under my care, on January 15th, 1896, suffering from typhoid fever. On the 6th of January she felt sick and had a headache, but did not lie up until the 8th, when owing to weakness she could not get about. Beyond having had pleurisy some twelve months previously, there was nothing to note in her previous history. As regards surroundings, a brother in the same camp was ill with typhoid fever. On admission patient complained of pain in the abdomen and the back, extending down the legs. The condition of the patient was as follows:—She was well built and well developed, with dusky flush over face, pupils dilated, lips fissured, sordes on teeth, tongue moist, but covered with a white fur.

Abdomen.—Tumid ; pain complained of over right iliac fossa ; splenic area increased upwards and to the middle line. Numerous pink, slightly elevated spots, disappearing on pressure, were present, mostly over the right half of the abdomen, also over the front and sides of the chest. The liver was slightly enlarged, but no pain was felt over the hepatic region. Bowels opened three times within two hours ; stools typically typhoidal. Heart sounds normal. Pulse regular, 96. Respirations 44. Râles at bases. Urine, sp. gr. 1030 ; no albumen. Temperature 104°.

Progress of the case.—From January 16th to 21st there was constant pain in the back and legs. The food was frequently rejected. Bowels opened six to nine times in the twenty-four hours. Nocturnal delirium was constant. Fresh crops of spots appeared on the abdomen, chest, left shoulder, forearms, and on the back. The temperature varied from 102° to 104·4°. Respirations were mostly 36 per minute. The alæ nasi moved freely in respiration. On the 21st very severe pain was complained of, situated over the right half of the epigastric region and over the right lower axillary and hypochondriac regions ; palpation over the latter region increased the pain. Respirations were now 40 per minute, and there was evidence of dry pleurisy over the right base, axillary, and lower mammary regions. Sickness and vomiting were constant, and the temperature rose to 105·8°. The day following (the 22nd) the conjunctivæ and skin of the upper part of the body were slightly jaundiced, bile was present in the urine, and the stools were frothy and white. On the 23rd the jaundice was much more marked, the pain over the regions before noted was still severe, and now extended around the umbilicus. The region of the gall-bladder was exquisitely tender. The next day the pain was so severe,—and stated to be general as well as local,—that the patient screamed when moved. The jaundice was still marked, and bile still present in the urine. Now for the first time both urine and fæces were passed unconsciously. The respira-

tions were increased to 50 per minute, and the breathing was *mainly abdominal*. There was now dry pleurisy on the *left* side as well as on the right. From this date the pain over the epigastric region was better, and ultimately disappeared; at times the patient vomited bile, *and the jaundice remained persistent from its first appearance (January 22nd) to the day of death on February 14th*, sometimes being more marked than at others. On one occasion, with the exception of the conjunctivæ being yellow it quite disappeared, but returned the day following as marked as before. Bile was almost constantly present in the urine. As regards the pulmonary complications, it must be added that on February 4th dyspnœa became extreme, the face cyanosed, the soft parts were drawn in, and the respirations numbered 56 per minute. The breathing was now *entirely abdominal*; the temperature, which had fallen somewhat suddenly, rose to $105\cdot4^{\circ}$; there was now evidence of pneumonia of the right base, with dry pleurisy over part of the right lung, and all over the left lung. From this time onward the dyspnœa was extreme, the respirations varied from 60 to 80, and eventually the patient died from the pulmonary complications.

As to other points of interest in the case, mention must be made of the—

(1) *Bowels*.—At the first onset of the jaundice the stools were white and frothy, but later on they became more coloured, and on three or four occasions bile was passed by the bowel and the stools were passed unconsciously.

(2) *Hæmorrhage* from the bowels occurred on the 25th of January, and was more or less constant till the 3rd of February, many of the stools consisting of pure blood.

(3) *Delirium* was constant throughout during the night, and frequent during the day.

(4) *Skin*.—On the 29th of January four pomphi appeared on the left leg, subsequently becoming bullous with dark reddish purple margins; at the same time

numerous pink petechiæ appeared on both legs. The bullæ, as also the petechiæ, subsequently disappeared, but the marks where the bullæ had been remained persistent and were distinct at the autopsy. On February 6th a swelling the size of a small Tangerine orange, purplish red in appearance, and extremely tender to the touch, appeared on the left hip. The skin over this showed no tendency to break. Subsequently this swelling entirely disappeared.

The temperature during the first few days varied from 102.2° to 104.4° . On the 21st January (the seventh day after admission) it rose to 105.8° in the evening. The ice-cradle was then used, and in the morning it was 101.4° . Three days later it rose to 105.2° , and two days after this to 105.4° . It then fell gradually, and on the 31st of January (the twenty-sixth day after first complaining of sickness and headache) registered 98.6° , then remained between 100.8° and 99° for four days, when it suddenly rose to 105.2° , and fluctuated between 101° and 104° for four days, then fell to 100.4° , and two days later to 99° . The day following it rose to 103.8° ; then fell to 97.4° ; then rose to 105° , fell to 99.8° , and the patient died within an hour.

Autopsy.—The body was greatly emaciated.

Thorax.—The *left lung* was adherent in all parts, the adhesions broke down fairly easily. The *right lung* was free over the lower lobe, and lymph was present over both pleural surfaces; this free space contained ten ounces of clear fluid. The anterior part of both lungs was markedly pale, and the lungs were in a collapsed condition. There was consolidation at the base of the right lung and a small area at the apex of the lower lobe of the left lung, the latter being in an earlier stage than the former. The cardiac walls were flabby, the right ventricle dilated, and the valves normal.

Abdomen.—The stomach was distended, its lower border reaching down nearly to the umbilicus; adhesions existed between the gall-bladder and the stomach. On reflecting the right half of the abdominal wall the gall-bladder was

found to be adherent to the peritoneum, and around the point of contact was a small circumscribed area of peritonitis. On gently breaking down the adhesions a perforation sufficiently large to admit a No. 10 catheter was noticed in the anterior wall of the gall-bladder, just above that part of the fundus which was adherent to the stomach. The gall-bladder extended for about one inch and a half below the anterior border of the liver; its contents, small in amount, were slightly purulent, its walls thin, a few small ulcers were seen at the fundus, and on the posterior surface was a small area so thin that it was almost perforated. In its neck was one solitary gall-stone resembling in its size, shape, and surfaces a mulberry. The commencement of the cystic duct was not blocked by the gall-stone, which was easily moveable. Unfortunately the hepatic ducts and the ductus communis choledochus were accidentally cut off during the removal of the specimen—the autopsy being performed under difficult circumstances. The liver itself was uniformly enlarged, and on section showed “cloudy swelling;” no bile exuded. There was typical ulceration of the intestine.

Microscopical examination of the liver showed nothing of importance. In the absence of any knowledge of the condition of the choledochus one cannot speak positively as to the cause of the jaundice; still the absence of any bile exuding from the liver on section, and the small amount within the gall-bladder, would lead one to think that the choledochus was not blocked to any appreciable extent; and it is certain that the gall-stone did not cause any obstruction; and had it blocked the cystic duct, that in itself would not produce jaundice if the hepatic duct and the choledochus were patent. A case of some interest in association with this is quoted by the late Dr. G. Budd.¹ A girl, aged 18, died from a severe attack of typhoid fever. At the autopsy fourteen gall-stones were found in the gall-bladder, and one completely blocked the cystic duct.

¹ ‘Diseases of the Liver,’ 1857, p. 197.

The gall-bladder was united to all the surrounding parts by lymph. No jaundice was present. One factor, however,—purulent inflammation within the gall-bladder—which is, according to A. Fraenkel,¹ capable of producing jaundice without any obstruction of the choledochus, was present in this case, and might have been the cause of the jaundice, and while one cannot associate this case in the same category as those of an infective nature consequent upon typhoid fever, pneumonic or relapsing fever, yet it may be claimed that the typhoid fever was the exciting cause, for it is well known that biliary calculi may remain for a long time and yet cause no symptom. A case in point presented itself the other day in a man aged 72, who died from renal disease. A single calculus resembling the one in the case now under review in its size, shape, &c., was found in the gall-bladder at the autopsy; no one symptom of such existed while the patient was under observation, and so far as the history of the case showed, never had done so.

Another point of much interest—not to speak of the age of the patient, eighteen years²—was the presence of local adhesions and perforation of the gall-bladder, which must be attributed, I think, to the gall-stone, for the discharge of such stones through fistulous openings in the abdominal parietes is not uncommon, though I cannot find a single instance recorded of such occurring during typhoid fever. Liebermeister³ cites two instances under his own observation of gall-stones rupturing through the gall-bladder and so causing fatal peritonitis.

This paper commenced with the consideration of the rarity of jaundice, clinically, in typhoid fever, and it must end by mentioning the rarity of perforation of the gall-bladder pathologically in typhoid. That the gall-bladder

¹ 'Centralblatt für innere Medicin,' No. 14, 4th April, 1896, p. 358.

² In an analysis of 395 cases collected by Hein, only 15 were under twenty-five, and only 3 under twenty. Quoted by Murchison, 'Diseases of Liver,' p. 382.

³ Ziemssen's 'Cyclopædia and Practice of Medicine,' vol. i, p. 154.

may be *ulcerated* in this fever has been shown by Jenner ;¹ it would, however, appear to be rare, for only one instance is cited in the 'Transactions' of the Pathological Society,² where Dr. Arthur Voelcker records the case of a man, aged 48, who died on the thirty-ninth day from perforation of intestine ; nine ulcers were found in the gall-bladder, "one of which, on the superior surface, extended into the liver." As regards *perforation* of the gall-bladder during typhoid, no case illustrative of this has been recorded in the 'Transactions,' and Wilks³ and Moxon state, "The gall-bladder . . . has even been found perforated ; we ourselves have never seen this." It would appear therefore, under any circumstances, to be rare. Murchison⁴ mentions one case ; a youth, 19 years of age, died on the fifteenth day of the fever from peritonitis, when the gall-bladder was found perforated. Barthez and Rilliet⁵ cite such an occurrence in a girl aged 12 ; and Mr. Monier Williams and Mr. Sheild record a case in the 'Lancet'⁶ occurring *after* typhoid fever.

In conclusion, it may be mentioned that suppuration of the gall-bladder may occur during typhoid fever. One such case is recorded in the 'Pathological Society's Transactions'⁷ by Dr. Hale White in a youth aged 17. At the autopsy there was general peritonitis around the gall-bladder, matting that organ and the lower edge of the liver and the intestines. The bile itself in this case appeared normal. There were neither gall-stones nor jaundice, the walls of the gall-bladder were very thin, and in some places almost perforated.

¹ 'Fever and Diphtheria,' p. 102.

² Vol. xlv, p. 79.

³ 'Pathological Anatomy,' p. 643.

⁴ 'Continued Fevers,' p. 566.

⁵ 'Traité Clinique et Pratique des Maladies des Enfants,' 3me édit., vol. ii, 1887, p. 612.

⁶ 'Lancet,' March 2nd, 1895.

⁷ Vol. xlii, p. 181.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 51.)

ON THE
PRESENCE OF THE TYPHOID BACILLI
IN THE URINE OF PATIENTS SUFFERING FROM
TYPHOID FEVER

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THE subject of this paper is one of considerable importance, and although in England it has been almost entirely neglected, it has already received some attention from bacteriologists on the Continent.

The first persons to work at the subject were Seitz (1) and Hüppe (2), who, in 1886, both published papers on the subject. They were followed in 1889 by Konjajeff (3), and in 1890 by Neumann (4) and by Karlinski (5), and in 1892 by Silvestrini (6), and lastly, in 1895, by Baart de la Faille (7) and by Dr. Wright (8), Professor of Pathology at Netley.

These observers all stated that in a certain percentage of cases, varying with each observer from $5\frac{1}{2}$ to 100 per

cent., they found the typhoid bacilli in the urine of patients suffering from typhoid fever. Neumann, for example, believed that he found them in eleven cases out of forty-eight examined, Karlinski in twenty-one out of forty-four; while Silvestrini examined seven cases and found them in every one, and in all stages of the disease, and this is practically also the result which Wright obtained, working so late as 1895. This observer examined seven cases, and found the bacilli "easily" in six,—finding them, indeed, in eight out of twelve examinations made. His cases were not examined during the first week, but he had no difficulty in finding the bacilli, which he regarded as typhoid, as early as he looked for them, namely, on the tenth and eleventh days of the disease.

Unfortunately, however, these results, which, if true, would be of extreme importance, cannot be accepted as of much value at the present day, for the simple reason that the observers quoted above had not the means which we now possess for distinguishing the typhoid bacillus from others resembling it, and especially from *Bacillus coli*, a bacillus which is to be found by no means uncommonly in urine. Most of the observers relied on the potato culture as their means of diagnosis, a method which is now entirely set aside, since we now know that many varieties of *coli* grow on potato in what used to be called the "typical typhoid manner."

De la Faille and Wright also, the two most modern observers, though using the three negative tests for typhoid,—the absence of gas formation in dextrose-gelatine cultures, the absence of the indol reaction, and the failure to clot milk,—did not stain the cilia of the bacilli which they regarded as typhoid, though this very important positive test had been introduced as far back as 1892. They did not also prove that their bacilli produced acid, thus failing to distinguish them from Petruchsky's *Bacillus fecalis alkaligenes*. (9) And lastly, they did not leave their milk and broth cultures for as long as a month before deciding whether no coagulation had oc-

curred, and whether no indol had been formed; and yet this is a matter of great importance if reliance is placed only on negative tests, for some varieties of *coli* only give these reactions, if indeed they ever give them, after being incubated for three weeks or even longer. They were unable also, of course, to use the serum test, since it had not then been introduced. It is evident, therefore, that we cannot accept as final any statements made by the observers who have so far worked at the subject. They found bacilli in the urine of typhoid patients which somewhat resembled typhoid bacilli; but whether or not they were so really we cannot say, for the simple reason that the tests applied were insufficient to determine this point. Judging from my own experiments, I should say that sometimes they really were true typhoid bacilli, but that very often they were not.

Considering the importance of the subject, and the fact that we now can with certainty distinguish the typhoid bacillus from others resembling it, I determined to investigate the matter, and to test in all available ways the bacilli which I might obtain, so that I might, once for all, settle the question *whether true typhoid bacilli do appear in the urine, and if so in what quantities, and at what periods of the disease.*

My method was as follows:—Typhoid cases were taken as early in the disease as they could be obtained, and the urine was then examined every second or third day until about a week after the temperature had fallen to normal. The urine was not, as a rule, drawn off by catheter, since it did not seem right to expose the patients to any possible risk, but it was passed direct into a sterilised flask. With one exception male cases only were used, to avoid any contamination from the vulva, and in the one exception, Case 2 of my series, the urine was drawn off by catheter, the orifice of the urethra being first carefully washed with soap and water, and then with carbolic lotion.

The urine having been thus obtained, it was examined as follows :

First, a drop was placed on a slide, allowed to dry, and then stained with gentian violet. It was then examined for bacilli under the microscope.

Secondly, gelatine plates were made. The gelatine was first poured out and allowed to solidify in the plate, and then the urine was dropped on to the surface of the gelatine and smeared all over it. In this way it was insured that all the colonies should be superficial ones—an important point, since a deep typhoid colony would certainly be missed. Two gelatine plates were always made, one containing as a rule one or two drops of urine, and the second about twenty. The exact number, however, of course depended on whether or not bacilli were found in the stained specimen, first made as explained above.

In the first four cases, I may add, some of the urine was set aside and left to incubate at 37° for forty-eight hours, after which plates were made with it. This is the method recommended by Professor Wright, by which he so often found bacilli, which he regarded as typhoid. I may say at once, however, that this method in my hands proved valueless, for *always*, whether the urine had been drawn off by catheter with every precaution, or passed direct into a sterilised vessel, or whether it was incubated alone, or after the addition of carbolic acid, *always* after forty-eight hours it became turbid, *swarmed* with micrococci, and was also often ammoniacal ; and I never, as a matter of fact, found the typhoid bacillus in the incubated-urine plates, when I had not already found them in the ordinary unincubated urine. I therefore gave up this additional method.

I felt, however, that even if I made a gelatine plate with twenty drops of urine (*i. e.* about $\frac{1}{2}$ c.c.), and failed to find in it colonies of typhoid bacilli, I was *not* therefore justified in saying that they were necessarily absent altogether from the urine, for the quantity of urine taken might have been too small. I therefore modified slightly

the method which Dr. Klein has invented for water analysis, and adapted it to the urine, and this method I used in the last three cases in addition to making plates with the ordinary urine. The apparatus employed was, of course, carefully sterilised before use.

The method consists in filtering by means of a force-pump about 200 c.c. of urine, which had been obtained as described above, through a Chamberland's bougie, then washing by means of a sterilised brush the micro-organisms and mucus left behind on the bougie into 3 or 4 c.c. of sterilised broth; from this two surface-gelatine plates were made, one with two, the other with fifteen drops of the washing: fifteen drops would correspond to about 750 drops, or about 25 c.c. of the pure urine; hence if no typhoid bacilli were found in a plate made with this large amount of urine, it seemed reasonable to conclude that they were really absent from the urine, and had not merely been missed from examining too small a quantity. [I may state also that it was impossible to take *more* than fifteen drops of the washing, for, as a rule, surface plates made with this quantity were thickly covered with micrococci, and had more been present it would have been impossible to detect a stray typhoid colony.]

I may at once here add that these filtering experiments simply confirmed the results obtained from the plates made with $\frac{1}{2}$ c.c. of urine. When the typhoid bacilli are not found by this method, they are also not found by the more tedious filtration method, for the simple reason that when the bacilli do occur in the urine they occur, as a matter of fact, in considerable numbers.

The plates having been made in the manner described above, they were set aside to incubate at 20°, and were examined after twenty-four, forty-eight, and seventy-two hours. If after this period no colonies resembling *B. coli* or *B. typhoid* had appeared, they were set aside. If, however, any suspicious colonies were seen, they were examined further as follows.

First, a trace of the colony was taken and examined under the microscope unstained. If the microbes proved to be bacilli, even although hardly any movement might be present, a shake culture in dextrose-gelatine and a stroke culture in ordinary gelatine were made. If after twenty-four hours gas had formed in the shake culture, the culture was set aside; if, however, none had formed, further cultures were made in broth and milk, and set aside for one month for the indol and coagulation tests. A culture was also made on agar, and after eighteen hours the motility of the bacilli was examined in a drop of broth. Also the cilia of the bacilli in this culture were stained by van Ermengem's method. Lastly, the serum test was applied.

Supposing, then, a bacillus obtained from the urine of a patient suffering from typhoid fever answered these tests—that is to say, proved to be a *bacillus*, *which often showed long threads, which possessed high motility, which was provided with eight to twelve or more cilia, which, however long kept, did not produce gas in gelatine or dextrose-gelatine shake cultures, while growing well in the substance of these media, which even after one month did not produce indol in broth cultures, nor clot milk, which, however, did produce slight acidity in milk after twenty-four hours, and which, lastly, gave a positive reaction when tested with typhoid serum,—then, and not till then, I think one is justified in saying that it is really the typhoid bacillus. Such has been the method which I have adopted in testing my bacilli, and all those which in this paper are called typhoid have answered correctly to these tests.*

I may add that *they were also pathogenic, for they killed guinea-pigs when injected into the peritoneal cavity, just like typhoid bacilli derived from other sources. Also they were decolourised by Gram's method.*

Results.—The results of my observations on the urine are as follows:—7 cases have been examined, and in all 61 observations have been made. In three cases I have

found the typhoid bacillus in the urine, and in four not. In no case have I found the bacillus before the beginning of the third week of the disease, although 17 observations were made during the first and second weeks. In one case the bacilli did not appear until the thirty-ninth day, although in this case ten observations had been made before this date. On some rare occasions the urine was rendered quite turbid by the numbers of bacilli excreted, but in others not so many were passed, though on eight occasions a drop of urine dried and stained showed the bacilli under the microscope. The urine containing bacilli was in two cases non-albuminous or contained but a cloud of albumen; in the third case, however, it was highly albuminous.

In more detail the results are as follows :

CASE 1.—James C—, aged 18, admitted into St. Bartholomew's Hospital under Dr. Hensley with typhoid fever on the eighth day of the disease. The patient had rather a severe attack. A certain number of rose spots were present, but the rash was *not* copious. Death occurred on the twenty-third day from perforation. A post-mortem showed the usual lesions of typhoid fever, together with perforation and peritonitis. The kidneys after death were engorged, but otherwise natural. The urine was examined first on the thirteenth day, but only micrococci were obtained on the plates. On the fifteenth day, however, typhoid bacilli were found in the drop of urine which had been dried, stained, and examined under the microscope. The number, however, was not very large. Cultures confirmed their presence. The urine was non-albuminous. On the seventeenth day exactly the same result was obtained. The urine was again non-albuminous. After this the number of typhoid bacilli decreased very much in number in the urine, and on the twenty-third day, *i. e.* the day of his death, only a very few colonies were obtained; they proved, however, to be still true typhoid

bacilli. The urine on this day contained a slight cloud of albumen.

CASE 2.—Ellen T—, aged 21, admitted into St. Bartholomew's Hospital under Dr. Gee on the seventh day of her illness. The urine in this case was drawn off by catheter, with full antiseptic precautions.

A very mild case of typhoid fever. The temperature reached normal by the fifteenth day; a few spots were present. The patient recovered without relapse.

The urine was examined on the ninth and twelfth days, and again two days after the temperature had reached normal for good, but no typhoid bacilli were ever found; micrococci were, however, always present, though the urine had been drawn off by catheter with full antiseptic precautions. And I may here mention that this is my invariable experience, namely, that however the urine is obtained, whether antiseptically by catheter or by being passed directly into a sterilised vessel, it always contains, even when unfiltered or incubated, a certain number of micrococci, which have probably come from the urethra. A few *B. coli* were also found in the urine of this case, one of which did not even after one month clot milk, nor give the indol reaction, nor bubble dextrose-gelatine. Its cilia were, however, those of *B. coli* and not those of *typhoid*.

CASE 3.—Frederick James R—, aged 18. In this case the urine was obtained antiseptically from the bladder on the post-mortem table, the patient having died about the twenty-eighth day, not from hæmorrhage or perforation, but apparently from the severity of the disease. Typhoid bacilli were found in the stained specimen, and many typical colonies were obtained on the plates made. The urine was highly albuminous.

At the commencement of his illness the patient had had a fair eruption of rose spots, but the number of spots was not in any way unusual.

Post-mortem.—The autopsy revealed typhoidal ulcera-

tion of the Peyer's patches, but the pure typhoid fever had become complicated by a secondary pyæmic infection. For, in addition to the intestinal lesions, recent endocarditis was present. The kidneys also were large, swollen, and deeply coloured, and showed many small infarcts; some of these latter had also broken down, and had formed small abscesses. The heart's blood was found by Dr. Kanthack to contain typhoid bacilli, but in addition proteus and streptococci. It is evident, therefore, one is not justified in attributing the kidney lesions to the unaided action of the typhoid bacillus.

CASE 4.—John S—, aged 15, admitted into St. Bartholomew's Hospital under Dr. Church on the sixth day of the disease. The patient had a severe attack of the disease, but recovered without relapse. The temperature reached normal by the twenty-first day. There were numerous spots. The urine was examined on the seventh, tenth, thirteenth, fifteenth, eighteenth, and twenty-first days, and also on the twenty-fourth day, that is three days after the temperature had reached normal, but no typhoid bacilli were ever found, only micrococci.

CASE 5.—Thomas P—, aged 18, the most interesting case of all. The patient was admitted into St. Bartholomew's Hospital under Dr. Lauder Brunton with a mild attack of typhoid fever. This ran its course, and the temperature reached normal by the twenty-fourth day. It almost immediately, however, began to rise again, and patient had a relapse, accompanied by a fresh eruption of spots. The latter, indeed, had been very numerous during the attack, and fresh crops kept coming out during the relapse. The temperature did not again reach normal until the forty-first day of his illness, after which it did not rise again. The urine was examined by the ordinary and by the filtration method on the following days:—the tenth, eleventh, thirteenth, sixteenth, nineteenth, twenty-second, twenty-fifth, twenty-seventh, thirty-first, and thirty-fifth

days of the disease, but no typhoid bacilli were ever found. Micrococci were, however, always present, and in the last five examinations a variable though small number of *coli*. Many of these on each day were examined with great care, but they all proved to be *coli*, not *typhoid*. I had by the thirty-fifth day almost given up hope; however, I went on with the case, and was rewarded by observing a most wonderful change which suddenly took place. My next examination was made on the thirty-ninth day of the disease, two days before the temperature reached normal for good at the end of the relapse. I at once noticed on obtaining the urine that whereas it had before been quite clear, on this day it was turbid; and on examining a specimen under the microscope I was astounded to find that it was literally swarming with bacilli. In short, the urine presented very much the appearance of a broth culture of typhoid turbid with growth. Cultures revealed the fact that the bacilli in the urine were, in fact, true typhoid bacilli.

The urine was examined again on the fortieth and forty-first days of the disease, the last being the day on which the temperature reached normal for good. On both of these occasions it was still turbid and contained typhoid bacilli in the stained drop, though not nearly in such large quantities as before.

The temperature had now reached normal for good, but still typhoid bacilli were excreted in the urine. Examinations were made three days, five days, eight days, ten days, thirteen days, seventeen days, and twenty-two days after the temperature became normal; but typhoid bacilli were always found, although in diminishing quantities, in the urine. After this date none were found, and the patient left the hospital. I should mention here that *while the bacilli were being passed the urine was acid, and contained either a faint cloud of albumen or none at all*. There was also never any cystitis.

The bacilli, when stained in the urine, as a rule seemed quite natural. On one occasion, however, they appeared

a little altered, taking the stain most irregularly. This, however, was only temporary, for the micro-organisms in the colonies obtained from them stained as usual, and presented all the characteristics of true typhoid bacilli.

CASE 6.—Alfred F—, æt. 28, admitted into St. Bartholomew's Hospital under Dr. Lauder Brunton. The case was a simple uncomplicated case of typhoid fever; some though not many spots were present. The temperature reached normal on the twenty-second day. There was no relapse.

The urine was examined by the ordinary method, and also by the filtration method on the following days:—the tenth, twelfth, fourteenth, seventeenth, nineteenth, and twenty-first day, and also two days, four days, and eight days after the temperature had reached normal, but no typhoid bacilli were ever found, only micrococci.

CASE 7.—George W—, æt. 19. The patient was admitted into St. Bartholomew's Hospital under Dr. Church. He had a mild attack of typhoid fever. A few spots only were present. The temperature, however, was rather late in coming down; it reached normal only on the twenty-seventh day. The patient then quite recovered without relapse.

The urine was examined on the ninth, eleventh, thirteenth, sixteenth, eighteenth, twentieth, twenty-fourth, and twenty-sixth days of the disease, and also when the temperature has been down two, four, and seven days, but on no occasion were typhoid bacilli found, although on all occasions micrococci were present.

Conclusions.—Such, then, are the results which I have obtained, and which I have arranged at the end in tabular form. These results, I think, justify the following conclusions:

(1) That true typhoid bacilli do occur in the urine in some, though not in all cases of typhoid fever.

(2) That when they do occur they are present in considerable numbers, so that a plate made with $\frac{1}{2}$ c.c. urine is amply sufficient to detect them; more laborious methods, such as the filtration method, being therefore unnecessary. Not uncommonly, also, if a drop of such a urine be dried and stained, the bacilli may at once be seen under the microscope. Occasionally, also, the urine may be rendered turbid by the enormous quantities of bacilli passed.

(3) My experiments also show that when the bacilli do occur they only appear towards the end of the disease,—that is to say, during the third week or later; their presence, therefore, can rarely, if ever, be an assistance in diagnosis. Observers who have discovered them as early as the third day have probably mistaken *B. coli* for them, or possibly Petruchsky's *Bacillus fæcalis alkaligenes*.

Lastly, in view of these facts, I should suggest that some antiseptic should always be added to the urine of typhoid patients as well as to their fæces, this precaution being taken at the present time, so far as I know, only with regard to the latter.

In conclusion, I should wish to thank Dr. Klein for having allowed me to work in his laboratory, and for the interest which he has throughout taken in my work. My thanks are also due to the physicians of St. Bartholomew's Hospital for having so kindly permitted me to make use of their cases.

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Day of disease on which the examination of the urine was made.	Urine, characters of.	Result given by a drop of urine dried, stained, and examined under microscope. 0 = no micro-organisms seen.	Plates made with 1 drop and $\frac{1}{4}$ c.c. of the fresh urine. 0 = no typhoid col. (some micrococci always present).	Plates made from urine incub. for 48 hours at 37°. 0 = no typhoid col. (micrococci col. always present).
CASE 1.—J. C.—, 18. Death from perforation on 23rd day; temp. never reached normal. Typhoid bacilli excreted from the 15th day onwards	13th 15th 17th 21st 23rd Clear, acid, no albumen idem idem Clear, acid, cloud of albumen	0 Many typhoid bacilli Many typhoid bacilli 0 0	0 Many typhoid col. Many typhoid col. 0 A few typhoid col.	0 Many typhoid col. Many typhoid col. 0 A few typhoid col.
CASE 2.—E. T.—, 21. A very mild case; temp. normal by 15th day. Recovery. Typhoid bacilli never found	9th 12th Temp. down 2 days Clear, acid, no albumen Clear, acid, no albumen Clear, acid and faint cloud of albumen Clear, acid, no albumen	0 0 0 0	0 0 0 (2 Coli colonies) 0	0 0 0 Many typhoid col.
CASE 3.—F. J. R.—, 18. Urine obtained antiseptically at the autopsy, v. text. Many typhoid bacilli found	28th Highly albuminous	Many typhoid bacilli	Many typhoid col.	Many typhoid col.
CASE 4.—J. S.—, 15. Severe but straightforward case; temp. reached normal on 21st day. Recovery. Typhoid bacilli never found	7th 10th 13th 15th 18th Clear, acid and faint cloud of albumen idem Clear, acid, no albumen idem Clear, neutral, no albumen	0 0 0 0 0 0	0 0 0 0 0 0	0 0 0 0 0 0

	Day of disease on which the examination of the urine was made.	Urine, characters of.	Result given by a drop of urine dried, stained, and examined under microscope. 0 = no micro-organisms seen.	Plates made with 1 drop and $\frac{1}{4}$ c.c. of the fresh urine. 0 = no typhoid coli. (some micrococci always present).	Plates made from the sediment left on bougie after filtration. 0 = no typhoid coli. (micrococci always present). - = method not employed.
CASE 6.—A. F.—, 28. Mild case; temp. reached normal on 22nd day. No relapse. Recovery. Typhoid bacilli never found	10th	Clear, acid, faintest trace of albumen	0	0	—
	12th	Clear, faintly alkaline, faintest trace of albumen	0	0	0
	14th	Clear, faintly alkaline, no albumen	One small mass of short thick bacilli, not typhoid	0	0 (2 cols. of short thick bacilli—not typhoid or Coli).
	17th	Faintly alkaline, no albumen, slightly turbid with phosphates	0	0	0
	19th	Clear, no albumen, faintly alkaline idem	0	0	0
	21st Temp. down 2 days	Clear, no albumen, neutral	0	0	0
	5 days	Faintly alkaline, no albumen, turbid with phosphates	0	0	0
	8 days	Slightly alkaline, no albumen, turbid with phosphates	0	0	0

CASE 7.—G. W.—, 19. A mild case, but temp. came down to normal only on the 27th day. No relapse. Recovery. Typhoid bacilli never found	9th	Clear, acid, no albumen	0	0	0
	11th	"	0	0	0
	13th	"	0	0	0
	16th	"	0	0	0
	18th	"	0	0	0
	20th	"	0	0	0
	24th	"	0	0	0
	26th	"	0	0	0
	Temp. down				
	2 days	"	0	0	0
	4 days	"	0	0	0
	7 days	"	0	0	0

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,'
Third Series, vol. ix, p. 55.)

DESCRIPTION OF PLATE XX.

On the Presence of the Typhoid Bacilli in the Urine of Patients suffering from Typhoid Fever (P. HORTON-SMITH).

FIG. 1.—The urine passed by Case 5 on the thirty-ninth day of the disease.

A drop of the urine was taken soon after it was passed, evaporated to dryness, and stained with gentian violet. $\times 400$.

Innumerable typhoid bacilli are seen in it.

FIG. 2.—From the same preparation as Fig. 1, but more highly magnified. $\times 1000$.

(Photographed by E. C. Bousfield, Esq.)

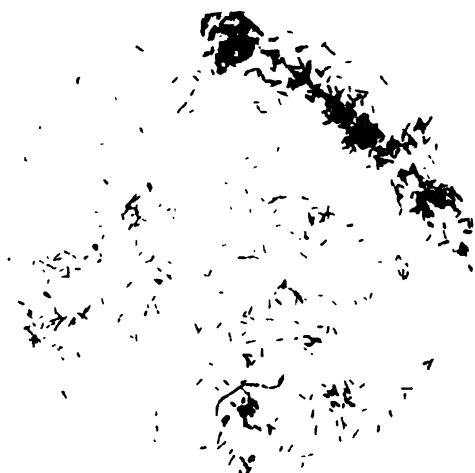


FIG. 1.



FIG. 2.



DESCRIPTION OF PLATE XXI.

On the Presence of the Typhoid Bacilli in the Urine of Patients suffering from Typhoid Fever (P. HORTON-SMITH).

FIG. 1.—The urine passed by Case 5 three days after the temperature had fallen to normal for good.

A drop of the urine was taken soon after it was passed, evaporated to dryness on a cover-glass, and stained with gentian violet. $\times 400$.

Many typhoid bacilli are still seen.

FIG. 2.—The urine passed by Case 5 eight days after the temperature had fallen to normal for good. Specimen prepared in the same way as Fig. 1. $\times 400$.

Many typhoid bacilli are still present.

(Photographed by E. C. Bousfield, Esq.)



FIG. 1.



FIG. 2



DESCRIPTION OF PLATE XXII.

On the Presence of the Typhoid Bacilli in the Urine of Patients suffering from Typhoid Fever (P. HORTON-SMITH).

FIG. 1.—The urine passed by Case 1 on the seventeenth day of the fever.

A drop of the urine was taken soon after it was passed, evaporated to dryness on a cover-glass, and stained with gentian violet. $\times 400$.

A fair number of typhoid bacilli are seen.

FIG. 2.—A specimen showing the cilia of the typhoid bacilli obtained from the urine of Case 3 (twenty-eighth day of the disease).

Prepared by Van Ermengem's method. $\times 1000$.

The cilia are typically typhoidal.

This specimen would represent equally well also the cilia of the typhoid bacilli obtained from Cases 1 and 5. In all cases the cilia were exactly like those represented in this figure, and were thus typically typhoidal.

(Photographed by E. C. Bousfield, Esq.)



FIG. 1.



FIG. 2.



ON SOME AFFECTIONS OF THE NERVOUS SYSTEM

MET WITH IN ASSOCIATION WITH AN
ATTACK OF

ENTERIC FEVER

BY

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I PROPOSE in this paper only to deal with a few of the complications or sequels of enteric fever due to affections of the nervous system. The occasional occurrence of *imbecility* as a sequel of enteric fever is well known, and is ascribed by Murchison to an anæmic or atrophied state of the brain. It has twice happened to me to see patients admitted into hospital in a state of apparent imbecility after an attack of enteric fever ; and as it is a sequel very rarely met with in patients treated in the hospital throughout the whole illness, I came to the conclusion that it was probable that the imbecility was mainly due to the insufficient feeding during the attack which home nursing

amongst the poor would be sure to entail, and this idea harmonises well with Murchison's view as to the pathology.

The following case seems to support this view. A boy aged 13 was admitted under my care at Charing Cross Hospital on October 14th, 1895, suffering from enteric fever, it being about the eighth day. The attack was a severe one, and for a week after he came in his temperature was almost continuously over 104° ; by the thirtieth day his morning temperature was normal, but the evening temperature was never quite satisfactory, and after eight days it began to rise again, and he had an exceedingly severe relapse, lasting four weeks, which was followed a week later by another relapse lasting five days. He was very delirious and noisy when he came in, and then passed into a state of complete mental dulness, taking no notice of anything that went on around him, but resenting any interference, and especially any attempt to feed him; from the day after his admission until November 15th he never spoke, and until a few days before this he showed no sign of understanding anything that was said to him, and he fought so against his food that it had been necessary to feed him through the nose. From October 19th to November 13th nasal feeding was employed, and for the greater part of this time he took absolutely nothing by the mouth; three feeds were given in the twenty-four hours; at first of half a pint each, but this was gradually increased to fifteen ounces. During the relapse he again ceased to speak, and became again apparently imbecile, and as troublesome about his food as before. Nasal feeding was resumed on November 22nd, and not finally abandoned until December 17th, and during this period we had the additional difficulty to contend with that he was sometimes sick just after a feed. His bowels were constipated throughout, and for many weeks he passed everything under him, and lay just where he was placed, with his thighs flexed on his abdomen; he objected to being disturbed at all, and seemed very tender everywhere. When he began to get a little better he gave

much trouble by biting and eating his sheets, a habit we only broke him of by leaving him between blankets. Just before Christmas, and before he was strong enough to sit up in bed, he begged to be allowed to draw and paint, and he would spend the whole day in his new-found occupation, hardly allowing himself to be interfered with even for food, and certainly not for so trivial an occurrence as the doctor's visit; the sister of the ward was quite distressed at not being able to correct what she considered his breach of good manners, and he did not recover his mental equilibrium till about the middle of January, when his mind was a complete blank as to everything that had passed, and his craze for drawing, in which he displayed very considerable talent, gradually waned, and finally ceased to exist. During this period all his hair came out.

On January 15th he complained that his left foot felt heavy; he had noticed numbness in it and pins and needles for some days, he said, and there was found to be decided foot-drop with increased knee-jerk and some ankle-clonus; subsequently the reaction of degeneration was found in the muscles of the tibialis anticus group, and the leg wasted a little; he was of course already extremely emaciated in consequence of his long illness. There was at no time any loss or alteration of sensation, and he never had any spontaneous pain in it, nor was there ever any muscular tenderness. He left the hospital on May 7th practically well.

There can be but little doubt, I think, that his mental condition was directly due to the difficulty in getting sufficient nourishment into him both during the original attack and during the relapse. At these times his face was quite expressionless, but he had some very slight intelligence, for he would put out his tongue when I came to his bedside; but if I asked him a question or told him to give me his hand, he would put his tongue out again, and his features showed no sign that he had comprehended what was said to him. When well he

proved to be a boy of considerable intelligence, decidedly above the average for his age. The paralysis coming on during convalescence was a decidedly interesting feature in the case. Cases of so-called peripheral neuritis as a sequel of enteric fever have been recorded by Dr. Handford in this country, and Dr. Osler in America; but it is an excessively rare sequel in so young a subject as my patient; though he had a good deal of general hyper-æsthesia he did not have the condition of tender toes which I think Handford ('Brain,' part 42) was the first to describe. My case may be compared with one recorded by Dr. Shore ('St. Barth. Hosp. Reports,' vol. xxiii). His patient was a married woman aged twenty-six; the paralysis affected the arms, and began about seven weeks after the fever was over. In my case the interval was shorter, but then my patient was much younger, so that it would appear possible that the rule which I believe holds good in diphtheria, viz. the older the patient the more is the onset of paralysis delayed, may also be true of enteric fever. I have once met with an instance of what I think might be called *kleptomania*. A boy aged eight, during early convalescence from enteric fever, stole a knife from the locker of the patient in the bed adjoining his, and on the following day sold it for a penny to another boy in the ward who had come to his bedside. On being accused of the theft he did not seem able to understand what all the fuss was about, and when he was well he seemed in all respects a very well-behaved boy, and the theft and subsequent sale were of such a barefaced nature that I had no doubt they were the acts of one who at the time was not fully responsible for his deeds. I may add that during the fever he had had an unusual degree of delirium, and he had shown some delusions.

Rigors may mean the supervention of peritonitis or the occurrence of perforation, but, as was pointed out by Dr. Gee ('St. Barth. Hosp. Reports,' vol. x), they may have no such grave significance. I have met with several in-

stances bearing out the truth of his observation. As a rule, when due to constipation or any form of simple intestinal irritation, they occur late in the course of the disease ; one patient under my care had two rigors, with an interval of one day between them, more than two months after her admission into the hospital ; the case had been one of great interest, as the patient had an attack of acute mania during the primary fever, but she had been quite convalescent for some time before the rigors. The temperature may run up very high ; I have known it to reach 106° , and subside almost as rapidly after the bowels have been relieved by enema. As a rule it may be said that the earlier the rigor occurs in the course of the disease, the greater is the probability of its being due to perforation or peritonitis ; but I have known a rigor on the twenty-second day of the illness to be dependent upon constipation. With the exception of the patient whose case I allude to directly, all the rigors of this kind I have met with have occurred in women, and Dr. Gee's patients were also women.

Dr. Osler has written the most complete paper on this subject that I have yet met with, in the ' Johns Hopkins Hospital Reports,' vol. v, but curiously enough he does not mention the possibility of constipation or intestinal irritation being a cause of rigors. In some of his cases the rigors appeared to be due to the injudicious use of some of the modern antipyretics ; of this group I have no personal experience, but in two of his cases the rigors were associated with venous thrombosis, and in this connection I might mention a case to which I shall allude more fully in the latter part of this paper. Suffice it here to say that my patient had eight rigors in the course of his illness ; the first occurred on the thirtieth day of his illness, and on the second day of the onset of his first relapse, at a time when, as Osler shows, rigors are not very uncommon. His second, third, fourth, and fifth rigors occurred during his second relapse on the sixty-eighth, sixty-ninth, seventieth, and seventy-first days of

his illness, and a few days after thrombosis had occurred in his left femoral vein, the right having been affected some ten days earlier. That the rigors were closely associated with the vein trouble was, I think, proved by the fact that during these few days a tender swelling about the middle of his left calf made its appearance, and exactly in the course of the short saphenous vein. His sixth rigor occurred on the seventy-fifth day, and was probably due to intestinal irritation; the patient ascribed it to his having remained a very long time on the slipper in the expectation that his bowels would be more completely relieved than they had been. The seventh and eighth rigors occurred on the eighty-first and eighty-seventh days respectively, and both were apparently due to mental worry, the first being associated with the fact that his night nurse had been taken ill, and he found himself in the hands of a stranger; and the latter occurred exactly one hour after we had told him, in response to a direct question, that the nurse was suffering from enteric fever. In each instance his temperature went up to 103° , and remained high for two days.

On two occasions I have met with *convulsions* in the course of enteric fever, and as this is not only a very rare occurrence, but the source of great alarm, I shall give the cases in some detail. Both cases were met with in private practice. The first occurred some few years ago in the person of a young married lady, aged twenty, whom I saw in consultation with Dr. J. Henry Philpot. She was confined of her first child on November 3rd, being attended by Dr. Gibbons, who has assured me that the labour was in all respects normal, and that she went on well until November 20th, when she complained of headache, and he took her temperature and found it to be 104° . She was at the time allowed to be on the couch every day. He ordered her back to bed, and as there was an entire absence of symptoms apart from the headache, the possibility of enteric fever was

present in his mind. On November 26th characteristic spots made their appearance, and he then transferred her to our care. Her temperature remained very high for more than the first week,—only once, in fact, failing to reach 104° . In the course of the second week there was marked diarrhoea, which was, however, controlled, and meteorism appeared early and was very pronounced, and very great difficulty was experienced in getting her to take her nourishment. On December 5th, at 10.45 p.m., she had a convulsion lasting two minutes, and later on that night at 1.30 a.m. she had a second. When I arrived I found that Dr. Philpot had already drawn off some urine and examined it, finding a trace of albumen, not more than about one thirtieth; her temperature at that time did not exceed 101° , and had not been affected by the convulsions. Her pulse was somewhat hard; the heart-sounds were clear, the first sound being especially sharp. We could arrive at no conclusion as to the cause of the convulsions, except that we did not regard them as uræmic. We gave her chloral hydrate gr. xx at once, and ordered a draught containing ammon. bromide gr. xxx to be given every three hours. Later on that day she seemed in no way the worse for the convulsions, nor could we discover anything that threw any light on their causation. She was very ill of course, but she had no really unfavorable symptom. She was taking her nourishment very badly, and was being partly fed by means of nutrient enemata, and she had for a day or two had retention of urine, necessitating the use of a catheter. The bowels were acting every day. Next day, at 3.15 p.m., i. e. about thirty-eight hours after the second convulsion, she had a third, after which the chloral was repeated, and at 9.20 p.m. she had a fourth, which lasted three minutes, and was considered by the nurses to have been the most severe of all, and after it her temperature went up to 104.8° . It was impossible to conceal the fact that the patient was now in a highly critical state. The attack had occurred at a most inopportune time, viz. before she had recovered from the

effects of the labour, and the attack had been from the outset decidedly severe ; and as we were entirely in the dark as to the causation of the convulsions, we felt that for aught we could tell they might be repeated at any moment. On the other hand, there was no sufficient reason for anything like despair. The patient was young, her previous health had always been excellent, and there was no tendency to any form of cerebral affection in her family. The albumen had disappeared from her urine, and in spite of the extreme difficulty in regard to nourishment, owing to her obstinately refusing to take it, her heart was acting well, the first sound being particularly good for a case of such severity. It seemed to us that the convulsions must be reflex. In infants, as is well known, convulsions are easily induced by a slight intestinal irritation. Our patient was in many respects reduced to the condition of an infant ; physically she was very weak,—indeed, she had no muscular power at all. The bowels were acting every day, the motions had always been submitted for our inspection, and we had considered them satisfactory. Still we decided to risk the effect of an enema, though the fear of re-inducing the diarrhœa, which at an earlier stage had been somewhat troublesome, was present to our minds. Accordingly, a glycerine enema was administered, with the result that a large, pultaceous, very offensive stool was evacuated. Her temperature came down to 101° that night, and the patient had no more convulsions.

The rest of the case I need not further describe, though our anxieties were by no means over with the cessation of the convulsions ; she had two recrudescences, and for many days was in a most critical state, but eventually she recovered,—thanks to her good constitution, and to most assiduous nursing, her evening temperature not becoming normal until the sixtieth day.

The second case occurred this summer (1896) in the person of a gentleman whom I saw in consultation with his medical adviser. I have already referred to this

patient when speaking of the occurrence of rigors. The patient was a German aged thirty-five, who contracted enteric fever whilst convalescing from his second attack of syphilitic myelitis. Nothing noteworthy occurred during the primary attack, which was of no great severity, and lasted about three weeks; in four days' time his temperature began to rise again, and on the second day of his relapse he had, as already mentioned, a rigor; the relapse, which was decidedly severe, lasted until the thirty-fifth day, and six days later, on August 14th, he had a convulsion. From his previous history I had fully, I may say almost confidently, expected that he would develop some paralytic trouble, so I was not surprised when on August 10th he complained that his left leg felt heavy; he had, however, no numbness or tingling in it, nor was there any affection of sensation, so we were able to reassure him as to the condition of his leg. Two days before this he had complained of some bladder irritation, *i. e.* frequency of micturition, but this had been completely relieved by a few doses of an acid mixture, the urine having been found to be alkaline. On August 14th he seemed to be going on well, but he had had some return of his bladder irritation, and his urine contained a little blood and some albumen; it was not, however, smoky. On the previous day he had complained of pain and tenderness in the lower part of the belly on the right side, about an inch and a half above the middle of Poupart's ligament; this had, however, disappeared after a hot fomentation. His temperature was normal, and his bowels were acting every day after an enema; throughout his whole illness he had always required an enema. At 6.10 p.m. this evening whilst on the slipper he had a convulsion, and was unconscious for six or seven minutes; his temperature did not go up. The motion was sufficiently copious, light in colour, partly formed, and moderately offensive. His doctor saw him at about 7.30, and found him very nervous about himself; his heart's action was very tumultuous, and his pulse could not be counted at the wrist. He passed

a very wretched night, but when I saw him on the afternoon of the following day his pulse had got back to its usual rate, which was about 120, and his heart-sounds were clear. Seeing the circumstances in which the convulsion occurred, and in view of my experience in the previous case, I had no hesitation in ascribing the convulsion to intestinal irritation, but I was nevertheless much puzzled at the extreme and persistent perturbation of the heart's action, which had lasted for more than twelve hours. During the next few days he frequently had the bladder irritation, and he also complained a good deal of pain at the old spot on the right side of the abdomen, but he complained most of all of pain in his penis, which for about six days was continuous, and kept him awake at night. There was nothing to be seen the matter with his penis; and as the patient was extremely nervous, we were disposed to think the symptom was of no importance, but his temperature had begun to rise a little, and was always between 99° and 100° until August 24th, when he complained that his right leg felt heavy, and on examination we found that it was swollen, and that there was tenderness over the femoral vein. This gave us a clue to his recent symptoms, as phlebitis of the internal pudic vein or of some of its branches would explain the pain and tenderness in his belly, the pain in the penis, the bladder irritation, and the presence of blood in the urine. It was necessary further to revise the opinion we had formed as to the cause of his convulsion, for the passage of a portion of thrombus to the heart gave us a much better explanation of all the symptoms at that time than the one we had been so willing to adopt. This date, August 24th, proved the starting-point of a second relapse of considerable severity, and very prolonged, for it lasted quite a month, and it was during this relapse that four of his rigors occurred, thrombosis of the femoral vein on the left side having supervened on September 5th. His evening temperature became finally normal on the ninetieth day of his illness.

In one of the six cases of convulsions in connection with enteric fever recorded by Murchison, they were associated with thrombosis of the left femoral vein, but it does not appear that he regarded the association as more than coincidental, and his attention seems to have been solely directed to the question whether the convulsions could or could not be attributed to uræmia. I believe that in the two cases I have reported I have succeeded in tracing the convulsions to their true causes.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 57.)

ON SOME POINTS
IN THE
SURGICAL PHYSIOLOGY OF THE FOOT

BY
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(COMMUNICATED BY MR. HOWARD MARSH)

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It is now nearly thirty years since an accident to one of my own feet specially directed my attention to foot-physiology. A subastragaloid dislocation inwards with displacement of the middle cuneiform bone upwards and of the cuboid downwards are all evident at the present time. The permanent shortening and somewhat flattened sole is shown by impressions made with printer's ink nine years ago (Fig. 1.) How the depression of my plantar arch was ultimately raised by exercises, in application of the bow-string theory which I had conceived, has been often told ;¹ first in a paper read before the local branch of the British Medical Association in 1874,² and expanded into a pamphlet in 1877, of which a copy is in the Society's library.³

¹ 'Lancet,' 1884, vol. i ; 'Brit. Med. Journ.,' 1888, vol. i.

² 'The Influence of Muscular Action in the Prevention and Cure of Flat-foot.'

³ 'On the Arch of the Foot.' It is noticed in the first edition of the 'Treatise on Surgery,' by Mr. Holmes. This, so far as I can discover, is the earliest account of the treatment of flat-foot by muscular exercise.

During all these years I have never had any occasion for doubting either the soundness of that theory or its general application to the treatment of acquired flat-foot. I maintain that as the plantar arch is developed by muscular action, so it may by the same agency be restored.¹ But while I am able to produce plenty of cases where complete



FIG. 1.—Shortening of right foot, permanently flattened, by subastragaloid dislocation and displacement of tarsal bones. Functional recovery. Author's own case.

recovery has been due to that agency, and to it alone, I have always felt, until recently, that I could not give a satisfactory answer to such a question as this:—If it be true that action of the long flexors (and especially of the flexor longus pollicis) forms the arch, how is it that excessive action, as in the case of dancers, does not over-form it? I have felt that such explanation as I have given in 'The Human Foot'² of the *limiting* as well as *forming* influence of some muscles was not so complete as it should be. We

¹ 'Edin. Med. Journ.,' January, 1890.

² Churchill.

know that excessive formation does sometimes occur; it is seen in the condition known as hollow or claw-foot (Fig. 2). The principal object of this paper is to demonstrate, with the help of a model, why these two features which give the alternative names are found together.



FIG. 2.—Hollow or claw foot. Characters: 1, claw-like toes; 2, prominent heads of metatarsal bones, with callosities on the sole; 3, exaggerated arch, most marked in middle line; 4, contraction of plantar fascia; 5, persistent equinus position; 6, attenuated calf. Compare Fig. 7.

Here we have, manifestly, perverted function of the toes, of the muscles acting upon them. In the most comprehensive book on anatomy that I know, and where one would most expect to find the natural action explained, I find this:—"The flexor and extensor muscles of the toes, including the lumbricales and the interossei, act like the corresponding muscles of the hand."¹ But this, while true if the toes be used as fingers, is altogether untrue and misleading if the toes be used as toes. Then the *flexors* do not flex; they are *pressors* of the toes against the ground

¹ Quain's Anatomy, vol. ii, part 2, 1892, p. 274.

(Figs. 3, 4). So, too, the extensors do not extend ; they are *tractors* acting from the toes, as fixed points, and drawing the moveable body forwards.¹ The two sets of muscles are antagonistic in the hand ; they co-operate in the foot. The

FIG. 3.

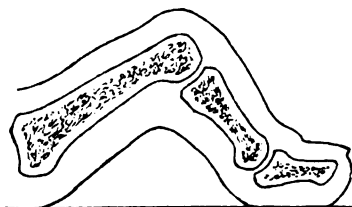


FIG. 4.



3.—Hammer-toe position, same as that of functional activity or pressure. 4.—Flexion.

pressing-down action of the flexors is necessary to give fixed points from which the extensors can act. Nor does this express the whole truth. The flexor longus pollicis, for instance, plays many parts at the same time.

(1) It presses the great toe against the ground, and so forms a point on to which the body can be lifted.

(2) In doing this, it assists in so lifting the body. In the model (Fig. 5) it is shown as the only agent in effecting the movement to, and sustaining the tiptoe position.

(3) It also draws the two extremities of the plantar arch towards each other, so relieving the strain upon the ligaments on the under side of the tarsus.

(4) By tending to include in the plantar arch everything between the final phalanx of the great toe in front, and the ankle behind, it lifts up the head of the metatarsal bone, which, as seen in the model, does not touch the ground line. Thus injurious pressure on that part is prevented ;

¹ This *tractor* influence, although disputed, admits of demonstration. Stand on one foot, the other uplifted and extended backwards as far as possible ; then spring forwards without lifting the heel from the ground. The movement is effected by the *tibialis anticus*, *peroneus tertius*, and the extensors acting, as can be seen, together.

the head of the bone is let down gently when the weight of the body overpowers the uplifting influence.

(5) Being attached to the inner side of the foot and to

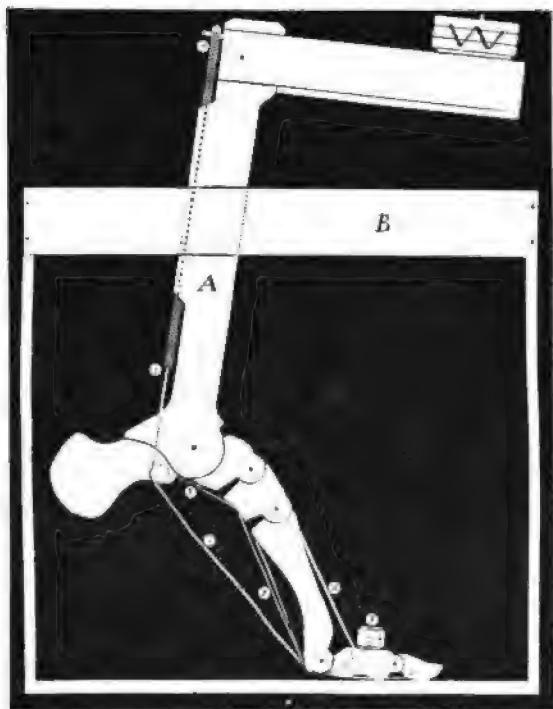


FIG. 5.—Model representing a foot, the bones united by hinges and balanced on the two phalanges of the great toe only, with the leg A (supporting the weight of the body, W) free to move between two parallel boards B. The great toe is kept straight and the curve of the plantar arch maintained by cords representing muscles in action. 1, 1, 1, 1 (elastic above), flexor longus pollicis; 2, divisions of tibialis posticus; 3, 3, flexor brevis and adductor pollicis, with a moveable bolt, 4, to set them free; 5, extensor.

the outer side of the leg, its action, by drawing towards a straight line between the two points of attachment, throws the ankle outwards. This serves a double purpose ; more

room is given for the opposite foot to pass by, and the weight of the body is thrown on to the outer side of the foot and off the inner side, where the weight falls when the sole is flat on the ground.

(6) It resists the tendency of the extensor pollicis to lift up the great toe as the latter muscle draws the body forwards.

(7) When the leg has been drawn forwards the distance between the two attachments of the flexor longus pollicis is increased; this muscle (already contracted) is then enabled, by a following-on or continued action, to propel the body onwards.

Sir James Paget, from whom it was my privilege to receive my first lessons in physiology, used to insist on the frequent duality of function. Here we have seven separate results from the action of one muscle.

But in order to have a straight toe as a fixed point from which this powerful muscle can efficiently act, it is necessary to have the co-operation of the flexor brevis pollicis, which holds down the first phalanx. The influence of the long flexor, acting alone, is seen by drawing on that muscle in an amputated foot; it flexes the inter-phalangeal joint, however firmly the foot be pressed downwards.

It is in this position that the great toe is represented in two striking pieces of sculpture to be seen side by side in the South Kensington Museum, the "Athlete strangling a Python" of the late Lord Leighton, and the "Teucer" of Mr. Thornycroft. It is also seen in the classic "Discobolus of Myron." But, *pace* the accomplished Professor of Anatomy at the Royal Academy, I say that it is not really artistic, because it is neither true to nature nor is it expressive of the highest ideal of the human foot, that of a pressing and not of a grasping organ.

Here I would say that the terms *origin* and *insertion* as applied to muscles are so misleading that I have often wished them to be abandoned. They suggest that muscles always act from their fleshy *origins* as fixed points, on their tendinous *insertions* as moveable ones. In the foot es-

pecially it is not so; either end may be the fixed point. I always try to speak of *attachments* only.

Now if the phalangeal attachment of the short flexor become a fixed point, it is clear that continued action of this muscle must *tend* to draw down the tarsal attachment at the other end. For reasons to be given, I hold that it does so act and effectively so.

Fig. 6 represents a scheme of the plantar arch which may be regarded as a construction of moveable pieces of bone held in position not only by ligaments, serving as braces, but also by muscles and tendons, serving as bow-strings or tie-rods. These are represented in the figure as cords. It is clear that if only one were present, as in the ordinary bow, and that one drawn too tightly, it must tend to over-form the arch. If, too, it remain firm, a weight placed over any point of the arch, as at W, sufficient to weigh it down at that point, must cause a corresponding

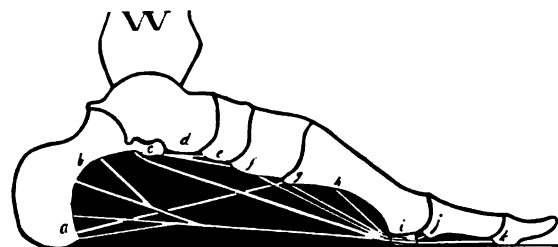


FIG. 6.—Scheme of plantar arch with muscles and tendons serving as tie-rods. *a b, j*, abductor pollicis and flexor brevis digitorum. *a, g*, peroneus longus. *c, k*, long flexors. *c, e f*, tibialis posticus. *f g, i*, flexor brevis and adductor pollicis. *h, j*, interossei.

elevation elsewhere. Here we have, however, other cords extending from one extremity, or a point beyond it, to the other extremity, or to intermediate points in the arch; while some go from one intermediate point to another. It is, I think, also clear that all these, if properly arranged and sufficiently strong, will combine to hold the arch in position and prevent either sinking or undue uprising.

A cord reaching from one extremity to the other could only be, in relation to the arch, a supporter or *formator*;

but most of the cords shown, while *formators* of that portion of the arch which they subtend, are controllers or, as I call them, *limitors* of the particular points in the arch to which they are attached, or beyond which they pass; they tend to *limit* any projection outwards at those points which might be caused by action of the *formators*. Now, of the muscles of and acting upon the foot, the *tibialis posticus* at the one end, and the *flexor brevis* with the *adductor pollicis* at the other, fulfil these conditions within that part of the arch which is most yielding, and of which the *flexor longus pollicis* forms the tie-rod or bow-string. In the model (Fig. 5) this is shown. It is true that the *flexor brevis* and *adductor pollicis* are not attached (as represented by the corresponding cords) to the base of the first metatarsal and cuneiform bones, but they are attached to parts of the tarsus opposite to those points.¹ I have, too, purposely omitted anything to represent the *peroneus longus*. The *interossei* are disregarded. I want to show that the muscles which I have represented in front (*flexor brevis* and *adductor pollicis*) are so placed that they would, if strong enough, be of themselves sufficient to hold down the arch and prevent undue formation. I do this because I allege that failure of these combined muscles to hold down the first phalanx is the cause of the claw-like toes seen in Fig. 2, and failure of them to hold down and limit the formation of the arch is the cause of the hollow foot which accompanies that condition. Moreover, failure to hold down the first phalanx leaves that bone free to rise, and so form an angle projecting downwards at the head of the metatarsal bone. Pressure at this point leads to callosities, another attendant condition, as seen in the photograph (Fig. 2). In health this is prevented by that which I have given as the fourth effect in action of the *flexor longus pollicis*.

¹ Objection is made that these muscles are not fairly represented by the cords, because of their extensive attachments to bone. But this, necessary in the foot, in order that the bones may preserve their shape, is not necessary in the wooden model where, as I contend, the essential conditions are fulfilled.

In the model it will be seen that the head of the metatarsal bone (Fig. 5) does not really rest on the ground line; the whole structure is balanced on the inter-phalangeal joint. It will be seen, too, that the great toe is straight, and the plantar arch moderately developed, clefts remaining open between the cuneiform and the bone on either side of it.

Now if I remove the bolt at 4 which retains the cords representing the flexor brevis and adductor pollicis, we find that, immediately, the head of the metatarsal bone goes down plump on the ground line; the great toe becomes flexed, and only wants the influence of the *extensor* to draw it backwards and give the complete claw-like character seen in Fig. 2; while at the same time the arch becomes much more pronounced, the clefts just mentioned disappearing (Fig. 7). Thus not only are the features which

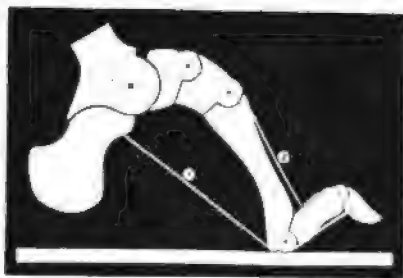


FIG. 7.—Showing effect on the great toe, on the head of the metatarsal bones, and on the plantar arch, caused by removal of the bolt, 4, in Fig. 5. Compare also Fig. 2.

give to this deformity the alternative names of hollow or claw-foot produced at once, but with them a third becomes manifest: the head of the metatarsal bone projects downwards, exactly as seen in the deformity (Fig. 2), causing the painful callosities on the sole associated with it. But why, in this deformity, is the exaggerated arch most marked in the middle line? Because it is in the middle line of the foot that the flexor brevis and adductor pollicis are attached, and these are the muscles to whose failure to control the arch the exaggeration is due; the peroneus longus which controls the inner margin does not fail. Other

features are also found—a persistent equinus position and an attenuated calf. Now if we suppose a growing boy to have boots too short for him, he finds that by walking on tiptoe he is more comfortable; he acquires the habit of relaxing his sole muscles, which, ceasing to act on the toes, allow the toes to contract, just as we find they do in an amputated foot when the long flexors are pulled. This contraction shortens the toes. The foot is also shortened by the bow-string or tie-rod action of the long flexor, third in the list I have given of the effects ensuing. Now although it is true that when the foot is frequently raised to the tiptoe position the calf muscles are developed, yet when the foot is persistently *held* in that position the calf muscles come to serve more and more as ligaments, less capable of relaxation at will. Then the muscles waste from want of *action* as muscles; hence the attenuated calf. The contracted plantar fascia is merely adaptive shortening.

In the latest edition of 'Erichsen's Surgery' (1895) it is said of this deformity that "the mode of production is very doubtful—the treatment is not satisfactory." The indications, as I see them, are to restore by operation the form of the foot so far as possible, and especially to correct fully the equinus position, which requires a very pronounced lengthening of the tendo Achillis—to the extent, it may be, of two inches. The more this is done, the more the toes are compelled to come into line with the metatarsal bones in order to reach the ground. This done, the indication is to restore function. The best exercises are those which throw the body backwards (when standing) until it is in danger of falling; then in the effort to recover position the flexor muscles must act as *pressors*, in order that the extensors may act as *tractors*. Thus the toes are brought into the fullest functional activity, and, paradox though it seem, we have the same exercise good for hollow or claw-foot as for flat-foot. But, indeed, the object is the same in both cases, to restore the functions of the toes, and so to restore the balance of forming and

limiting agencies on the plantar arch, of *formator* and *limitor* muscles. It does not, however, follow that forces sufficient to prevent over-formation, are sufficient to correct it.

I have now to explain why, in the stage dancer, the plantar arch is rather flattened than over-formed. The sole muscles combining to act as flexors of the first phalanx are specially needed to keep the great toe in a straight line, and two of these muscles are, as I have shown, also *limitors* of the arch. The *limitors* counteract, or more than counteract, the excessive formator influence of the long flexors. Moreover, the fleshy mass of hypertrophied sole muscle fills up the hollow of the sole, and increases the appearance of flattening.

Here let me point out that the same muscles may serve in both capacities. I have demonstrated on the model that removal of the cords which imitate the flexor brevis and adductor muscles results in exaggeration of the arch. But the formator effect of an elastic cord imitating the long flexor was then in full operation. Now, having restored everything (as in Fig. 5), if I remove this latter agency the arch still stands and supports the weight as before, although not so firmly. But if now I remove the bolt at 4, just as I did on the former occasion, the arch collapses altogether, showing clearly that the cords which served as *limitors* of the arch on the first occasion were serving as *formators* or supporters on the second.

To sum up: hollow or claw-foot is due to persistent activity of the *formator* agency (long flexor) with failure of *limitor* agency (short flexor and adductor), while flat-foot is due to failure of muscular action altogether.

The many and varied and contradictory views which are still given as explaining this latter condition involve, as it seems to me, something like a scandal in a scientific profession, having regard to how common the deformity is. A recent writer says, "The muscles, tendons, ligaments, bones, fascia, and even an improper mechanical construction, have each been considered the chief factors in its

production.¹ To this list might have been added rickets (Prof. Ogston²) and "immoral practices" (Mr. Clement Lucas³). But to me equally astonishing is it to find those, who admit muscular action as an agent in supporting the plantar arch, so contradictory in their views. Here we find paralysis of a particular muscle assigned as a cause; there the division of its tendon is recommended as a remedy. Nor is the special importance of those which I regard as the chief agents anywhere recognised. I cannot recall a single writer on the subject who mentions those which I have so long regarded as bow-strings or tie-rods—the long flexors. In a very recent book on



FIG. 8.—Slight curve of the plantar arch with the sole resting on the ground.

anatomy, edited by one distinguished surgeon (Mr. Henry Morris), we are told by another (Mr. Jacobson) that the arch is maintained by (1) plantar fascia, (2 and 3) calcaneo-scaphoid and calcaneo-cuboid ligaments, (4) tibialis posticus, (5) peroneus longus, (6) tibialis anticus; there is no mention of the long flexors: while a recent French writer, who does me the honour of discussing my views, mentions the flexor longus pollicis only to say that it cannot have

¹ James K. Young, 'Orthopædic Surgery,' 1895.

² 'Brit. Med. Journ.,' 1888, vol. ii.

³ Ibid., 1884, vol. i; 'Lancet,' 1892, vol. i.

any influence on the plantar arch,—“ne saurait avoir d'influence sur la voûte plantaire.”¹

Figs. 8 and 9 show the effect on the plantar arch of the tiptoe movement; they are taken from photographs. If the manifest increase in the curve be not due to the bow-string or tie-rod influence of the long flexors, acting as I have demonstrated in the model, I think that I may fairly ask to what is it due? It proves a shortening; but Camper taught that the foot, in this change of position, *lengthens* one inch. This statement, as a fact, is still made.²



FIG. 9.—Increase of curve of the plantar arch in the tiptoe position. Compare Fig. 8.

In cases of flat-foot where the deformity of the bones is such that the plantar arch cannot be replaced, the probability, or even the possibility of cure, by muscular action alone, has been questioned. My answer may be stated thus:

“A persistent effort to conceal a deformity tends to cure the deformity.” This, one of the many expressions for which I am indebted to my old teacher, Sir James Paget, is but another way of saying that the body tends to retain

¹ ‘Traité de Chirurgie,’ Duplay and Reclus; Masson, éditeur, 1892.

² Parkes’ ‘Manual of Practical Hygiene.’

the form into which it is vigorously and persistently placed, which is obviously the more effective in proportion as it is done in opposition to resistance. We know that muscles developed by use tend to remain taut and firm when not in use. We know that continuous pressure, such as a muscle in a state of tonic contraction would exercise on the surface of a joint, tends to cause wasting, and so to make the pressure even throughout the joint surfaces. The whole theory and practice of physical education, as I understand it, is based on these laws. The body is maintained in position by the exercise of its function—to move. The agency which moves, supports. The muscles which *by* their action move, *in* their action sustain, and so relieve tension of ligaments. Professor Ogston, who seeks to explain why “writers have deceived themselves regarding their cures for flat-foot,” rejects “gymnastic exercises” and other “proposed cures.”¹ But even he states that “the normal shape of the bones is admittedly produced not so much by forces within themselves as by normally acting muscles and such like agencies.” With “static deformity,” as due to want of muscular support, we are familiar. Of “labour deformity,” of the injurious influence on the form of excessive muscular action, we also something know. I ask, then, is it probable that muscles are potent to develop the form of the body, potent to maintain the form, potent also to deform, and yet impotent to re-form it? Let me point out, too, that in the treatment of flat-foot we have the advantage of ordinary exercise of the functions of the feet. Every step taken in good walking promotes the cure. The foot is put into a good position against the resistance due to the weight of the body. Surely, then, the success to which I have so often testified is not a matter for incredulity. Let me give one case. Two and a half years ago Mr. A. C. Fletcher, of Charterhouse Square, asked me to take charge of the son of a friend of his living here in Gloucester. The patient was apprentice to a yacht-builder on the coast,

¹ ‘Brit. Med. Journ.,’ 1888, vol. ii, p. 1148; 1889, vol. i, p. 293.

and was shown by Mr. Fletcher to a very old friend of mine, Mr. Howard Marsh. It was agreed that the worse of the two feet should be put up in plaster of Paris and the case sent down to be under my care. How very crippled this young man then was, these surgeons can testify. In spite of all the difficulty due to his occupation he has recovered, and long ago ceased to have any trouble with his feet, and this as a result of directed exercise without other treatment.

There are, however, eminent surgeons who write on flat-foot and who ignore muscular action, or want of it, as at all concerned. Sir William Stokes, for instance, does not include it in the remedies for even slight cases.¹ He gives a drawing of the bones of a flat-foot taken from "a powerfully built muscular individual," where "no evidence existed of atrophy of any muscle." How it could be possible for the bones of the foot to rest on the ground line and yet not have caused atrophy of the sole muscles; and how the long flexors could have continued to act on a foot in that condition without the sole muscles to co-operate with them, are questions which at the time of the report I raised in vain.² Flat-foot, as I insist, cannot be or, being, cannot continue with active long flexor muscles.

To promote such activity under the most favorable physiological conditions ought to be the surgeon's aim, not only in flat-foot, but also in other painful affections.

In order to understand how troubles result from defects of function, or, in other words, from wrongly directed movements, it is necessary to realise the position of rest,³ for the foot as a whole and for the toes. This, as in other parts of the body, is the same in fatigue and in pain. It is that of the least tension of ligamentous structures, least pressure on articular surfaces. The point involves ques-

¹ 'Brit. Med. Journ.,' 1894, vol. ii.

² Ibid., 1894, vol. ii.

³ "The Position of Rest in Fatigue and in Pain," 'Brit. Med. Journ.,' 1878, vol. i.

tions of so much surgical importance that I wish to call attention to the plaster cast from which the frontispiece to 'The Human Foot' is taken (Fig. 10). First, the foot is extended and inverted, just as seen in anyone suffering pain from a kick on the shin and hopping on one leg, or lying on his side in bed. The degree of extension, as I

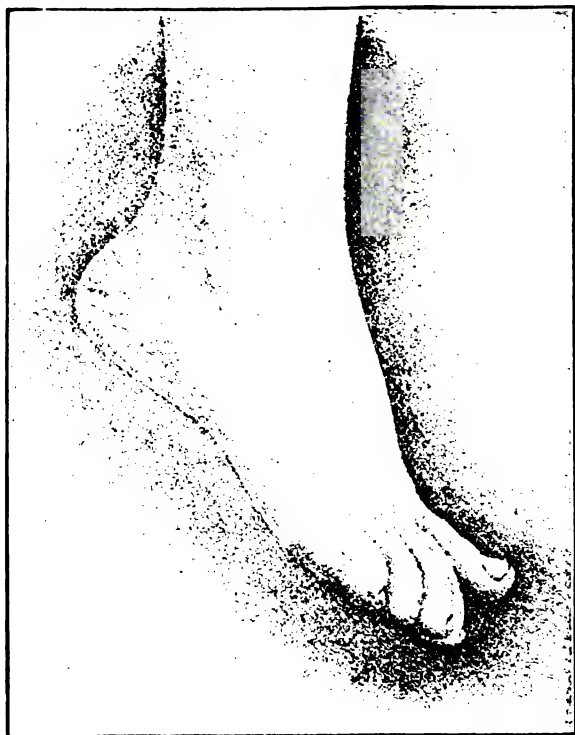


FIG. 10.—Position of rest. Extension and inversion with great toe directed over the second.

find it, suggests the necessity for a greater angle than is usually given for fixation splints or apparatus. Mr. Arbuthnot Lane regards abduction or eversion as the position of rest; but although, as he states, the feet do turn outwards when the body is laid on the back, that is

due to the weight of the thighs. Let anyone do as I did on reading Mr. Lane's article,¹—lie on his back, fasten a strong strap round the pelvis, and then stuff rolled-up bandages behind the trochanters until they are completely supported. I found then that the inner margins of the feet came into apposition throughout. As the foot hangs from the leg in the standing position it is, in relation to the leg, inverted. This, as I contend, is the position in which the footfall—it ought not to be a foot-placing—does in physiological walking come to the ground. That 824 cases of blistered feet occurred on the march to the New Forest manœuvres last year is not creditable to military hygiene.² But such things will be until the military march has been reformed.³ They need not be. To give a proper hold of the ground and so prevent friction, and at the same time to give best support to the plantar arch, and generally the best physiological conditions, the feet must not be everted.⁴

Surely it is better that the body be drawn forward *along* the line of the foot, of the muscles acting upon it, and *across* the hinges upon which the foot moves, rather than obliquely *across* the line of the foot and *along* the line of the hinges.

The position of rest for the great toe, extended and lying somewhat over the second, and its plane of movement, obliquely downwards and inwards away from the others, are points of great surgical importance; but I have not seen them noticed. If the great toe be fixed in the position shown, as it is generally fixed in the median-pointed boot, then such movement as it can make is in the natural plane, and when it is still it is really at rest. If,

¹ "Fallacy of the Vertical Foot-piece," 'Brit. Med. Journ.,' 1893, vol. ii, p. 1049.

² See Report of H.R.H. the Duke of Connaught on these manœuvres in 1895.

³ The arguments in favour of this reform, as set forth in 'The Human Foot,' are fully accepted by Mr. Walsham ('Deformities of the Foot,' 1895). The present practice is defended in Parkes' 'Hygiene.'

⁴ 'Lancet,' 1884, vol. i.

however, it happen to be packed beneath the second toe, then if it move at all it must be in a plane for which the joints are not adapted (Figs. 11, 12). Grinding of the cartilage and consequent irritation is a necessary result. Then the condition known as "hallux flexus," "hallux rigidus," or "hallux dolorosus,"¹ in varying degree of severity, is liable to follow. The toe is flexed because it is held down beneath the second; it is rigid because movement is

FIG. 11.



FIG. 12.



11.—Great toe packed over the second, its position of rest, conducive to hammer-toe. Compare Fig. 3 and Fig. 10. 12.—Great toe packed under the second, conducive to hallux flexus or rigidus.

painful; it is painful more or less, even when still, because fixed in an unnatural position; it is never in a position of rest. If sufficient muscular inactivity be attained, flat-foot also ensues.² But when the great toe is fixed in the former position on the top of the final phalanx of the second toe, it lies there on a sort of bed formed by the final phalanx which in the so-called *flexion* of the toe lies flat on the ground, while there is an elevation of the joint between the first and second. The toe being held in this position by the great toe and straightening prevented, a hammer-toe results (Fig. 3). In any case, but the more important in the hallux flexus position, to set free the great

¹ "Deformities of Great Toe," 'Brit. Med. Journ.,' 1887, vol. i; Walsham and Hughes, 'Deformities of the Human Foot,' 1895.

² I can recall three instances where footprints have been given as illustrative of flat-foot where the impress of four toes only has indicated this condition: Hare, 'Lancet,' 1889, vol. ii, p. 953; Walsham, *op. cit.*, fig. 230; also figs. 239, 247, 251; Tubby, 'Deformities,' 1896, p. 465.

toe is clearly indicated. A boot with a straight inside line is obviously needed. More difficult, as I find, is it to get patients or surgeons to recognise the need of socks or stockings which have either a corresponding shape or a separate stall for the great toe : the pointed sock, tightened by friction against the side of the boot when the foot is inserted therein, effectually holds the toes in the false position.

I dissent entirely from the statements of Sir G. Humphry that "the toes are shut up in leather and not used,"¹ and that "their services can be spared without detriment to the rest of the frame."² It is not so. They are generally used, and as the smaller ones move in a vertical plane there is no reason why they should not move together, even in a tight boot. These toes are necessary if only to provide for their corresponding metatarsal bones the protection from pressure on the ground which, as I have shown in the model, is served by proper use of the great toe. To the want of this influence are due the callosities on the sole so often found. When the toes are used the skin is free from anything of the kind ; the rugæ of the skin are distinctly seen in the printer's-ink impression shown in Fig. 1. To want of this influence are also due the conditions known as "Anterior metatarsalgia,"³ "Morton's toe," and the unnamed sufferings in the same region from time to time described.⁴ They are all evidences of defective function. Nor can the bow-string or tie-rod influence of the flexor longus digitorum on the plantar arch be properly exercised unless the smaller toes be efficient. Show me the condition of the toes in any foot, let me see how their functions are exercised, and I will predict the condition of the plantar arch.

Toes which overlap each other cannot take such good

¹ 'Lancet,' 1894, vol. i.

² 'Human Skeleton.'

³ 'Lancet,' 1889, vol. i, p. 437. Dr. Auguste Pollosson, of Lyons, in describing this condition, states that the pain is more likely to occur in going down than uphill. Exactly so ; the toes are more used in going uphill.

⁴ Guthrie, 'Lancet,' 1892, vol. i.

bearing on which to act as they do when each presses on the ground line. When the extensors act on toes not firmly fixed by pressure downwards, they draw them upwards, with resulting friction and corns.¹ And, indeed, in almost any defective condition of the foot there is a surgical indication to correct or renew the function of the toes, to compel each of them by active use to, literally, find its level.

These are some of the grounds on which I base my plea for a better recognition of foot-physiology as a subject intensely interesting from a philosophic point of view, and as having important relations to surgical practice.

¹ All the toes, when at rest, should be straight. The semi-flexed position, sometimes described as if it were normal, is not really so.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 85).

ON THE CLINICAL BEARING
OF
SOME EXPERIMENTS ON PERITONEAL
INFECTIONS

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No doubt much of the *experimental* work which has been done in the past will be utilised *clinically* in the future. Although at present we are unable to apply results derived from the laboratory to the welfare of our patients, yet it is of importance that we should avail ourselves as far as possible of this great means of adding to our powers of diagnosis, and more especially of treatment.

No apology is therefore necessary for calling your attention to the experimental results which I propose to lay before you, since they appear to have a distinct bearing on the treatment of, and on the safeguard from infections of the peritoneum.

The first point to be raised is in regard to a means of prophylaxis in cases where there is some probability, or at any rate some possibility, of unavoidable contamination of the peritoneal cavity if a trans-peritoneal operation is

undertaken (such as abscesses of typhlitic or pyosalpingitic origin).

In 1894 Dr. Issaëff,¹ a bacteriologist, published in Germany a remarkable series of experiments upon peritoneal infections which arose in connection with investigations upon the cholera vibrios.

So far as I am aware, the results of these observations on animals have never been suggested as available for use upon man.

He showed that several different substances, when injected into the peritoneal cavity of guinea-pigs, were capable of affording protection to the animals which were inoculated with cholera vibrios upon the following day. By means of the preliminary injections an increase of the "power of resistance of the part" is established, so that an otherwise fatal dose of microbes can be tolerated; this condition is attained in about twenty-four hours, and lasts about four days. It is to be observed that this condition of exalted general resistance has nothing to do with specific or special immunity; an animal protected by means of a previous injection of simple saline solution is rendered refractory not only to cholera and other vibrios, but also to other pathogenic organisms (*Bacillus typhi abdominalis*, *B. coli*, *B. prodigiosus*, &c.). It also differs in its transient nature; specific immunity gives a more intense and more lasting protection against the specific microbe.

Many writers on immunity have failed to take cognizance of the difference between these conditions of "local general resistance" and "specific immunity," so that often their deductions have to be weighed with caution in regard to the complex question of immunity.

Issaëff's experiments were made with a variety of different kinds of vibrio, after previous treatment with a number of different substances. I may be permitted to quote the following list from his paper, the doses in each case being 1 c.c. given twenty-four hours before infection

¹ 'Zeitschrift f. Hygiene u. Infektionskrankh.,' xvi, 1894, p. 287.

with the cholera vibrios. The animals were guinea-pigs of about 200-gramme weight.

Nature of previous treatment.	Maximal non-fatal dose.	Multiple of max. non-fatal dose for normal animal.
0. Normal . . .	$\frac{1}{18}$ loop ¹	—
Water . . .	$\frac{1}{3}$ loop	=5
Urine (human) . . .	$\frac{1}{3}$ loop	=7.5
Bouillon (pepton) . . .	$\frac{2}{3}$ loop	=10
Serum (human) . . .	$\frac{2}{3}$ loop	=12
Nucleinic acid (2 per cent.) . . .	1 loop	=15
Tuberculin . . .	1 loop	=15
Potent cholera serum . . .	4 loops	=60

It will be observed how very considerable is the effect when the special or specific serum from an animal immunised against the cholera vibrio was employed. Here two factors were in evidence:—1, the increased general resistance; 2, the specific action of the cholera serum.

In the course of my experiments several different kinds of microbes have been used.

The following is an instance where typhoid bacilli were the infecting agent, and rabbits were the animals, the preliminary doses (1 c.c.) being given twenty-four hours before infection.

No.	Previous treatment.	Infected with	Result.
1 . . .	0 . . .	4 loops .	Died in under 17 hours.
2 . . .	0 . . .	3 loops .	Died in 9 days 22 hours.
3 . . .	1 per cent. NaCl	4 loops .	Recovered after illness.
4 . . .	Typhoid serum (horse)	6 loops .	Recovered; hardly ill.
5 . . .	"	9 loops .	"

In other experiments guinea-pigs have been protected by sterile injections of nutrient broth against *B. coli*, *B. pyocyaneus*, and *B. prodigiosus*, as well as the above-mentioned vibrios and typhoid bacillus.

I do not propose to enter here into the anatomical details of the nature of this condition of increased resistance; let it suffice to say that the bactericidal power of the peritoneal fluid is greater than that of normal peritoneal fluid;

¹ 1 loop = about 2 mgrm.

and also that it contains a very large number of active phagocytes of two kinds,¹ which are not seen in normal peritoneal fluid.

These two facts, viz. increased bactericidal power of the fluid and increased number of phagocytes, together account for the protection which is given; supplemented by the specific substances of immunised sera when such have been administered.

In view of these facts it ought to be earnestly considered whether some similar preliminary treatment should be undertaken before operating through the peritoneal cavity upon abscesses.

In some cases contamination of the peritoneum can be avoided by operating in two stages (Sonnenburg, in appendicitis); but this is not always possible.

Two points deserve further consideration and attention. First of all, be it observed that the protection which can be afforded is *not unlimited*; it therefore follows that scrupulous care must be taken in order to make the unavoidable contamination minimal in amount,—in fact, as if no protective agency had been called into requisition.

Secondly, we have to discuss the nature of the injection which it would be profitable to employ in any given case. Here let it be understood that any suggestions on this score must be regarded as tentative, for as yet we have no actual experience in the matter in man. The point to be aimed at is TO PRODUCE AN AMPLE LEUCOCYTOSIS IN THE PERITONEAL FLUID, AND ALSO IF POSSIBLE TO GIVE A SUPERADDED SPECIFIC PROTECTION BY AN ASEPTIC INTRA-PERITONEAL INJECTION INTRODUCED ABOUT TWENTY-FOUR HOURS BEFORE PROCEEDING TO OPERATE.

If it were possible to know exactly what noxious microbes would be encountered in any given case, the obvious precaution to take would be to give a previous injection of the serum of an animal immunised against the

¹ Finely granular oxyphil or polynuclear leucocytes or microcytes, and macrophages.—*I*de 'Proc. Brit. Assoc.,' 1896, September 23rd; and 'Journal of Pathology,' March, 1897. (This has just come out.)

same microbe and its toxin.¹ Unfortunately we are not able to gain exact knowledge upon this point until the time of operation, and we have to rely upon more or less shrewd guesses. It becomes a matter of some importance to know *what microbes will probably be encountered*, and to guide our steps according to this probability.

To this end, I have made a number of observations upon fatal cases of peritonitis which have occurred from time to time in Guy's Hospital.

Before discussing these cases a few words may be said upon the literature of the subject.

Much evidence has been adduced to show that peritonitides and abscesses arising during the puerperium or after abortions are to be ascribed to streptococcal infection.² It would therefore be advisable, in cases of abscess having such origin, to protect the peritoneum with anti-streptococcic serum the day before proceeding to open the abdominal cavity.

Cases of cœliotomy in which a healthy peritoneum has been contaminated by the surgeon have often been shown to have been infected with staphylococci or streptococci, though in some cases other organisms have been found. However, cases of this nature are not strictly included in the class under consideration; and as far as they are concerned it can only be said that if a surgeon and his assistants, for whom he is responsible, are unable to be sufficiently cleanly, they had better desist from undertaking these as well as other operations.

In recent years great stress has been laid upon the importance of *B. coli communis* as the prime ætiological factor in cases of intestinal origin. Some observers,³ indeed, have gone so far as to use carbolised culture media (in which

¹ Anti-bacterial sera have no antitoxic action as a rule; antitoxic sera have both antitoxic and anti-bacterial action (*vide, e. g.*, Wasserman, 'Zeitschr. f. Hygiene,' 1896, vol. xxii, p. 263).

² *Vide* Hahn, &c.

³ *Vide* Malvoz; and again Laruelle, who examined two cases resulting from strangulated hernia, one of them five days after death (!); also Roswell Park.

the *B. coli* is one of the few organisms capable of growing) in order to prove their point. As a matter of fact, the difficulty is to get the other organisms and not *B. coli* to grow.

E. Fränkel (fifteen cases) and Predöhl (fourteen cases) tend to lay stress upon the presence of streptococci in these conditions.

Tavel and Lanz, who are also no enthusiastic supporters of *B. coli* as the essential factor, report twenty-eight cases with streptococci out of a total of forty-one (= 68 per cent.).

Max Oker Blom, in studying the penetration of the gut wall after experimental strangulations, remarks that various cocci were present besides *B. coli*.

Barbacci found *B. coli* in all the fourteen cases of peritonitis of intestinal origin that he examined, yet he is very careful to insist upon the fact that "although it may be the only cultivable microbe present, it cannot be regarded in the character of a truly pathogenic agent in the ætiology of perforative peritonitis."

A. Fränkel says that even with a perforated gut the peritoneum may contain pure cultures of *B. coli*; this is not in accordance with my experience, as will be seen in the record given below,—in fact, pure infection with *B. coli* appears to be a rare condition when the peritoneum is thoroughly examined, and suitable nutrient media used. I have often obtained pure gelatine plates of *B. coli* from cases in which other cultivating media yielded abundant cocci, and other forms as well.

There is much evidence that *B. coli* is pathogenic for man (abscesses, empyemata, &c.); and I am far from denying that it may have a rôle in certain cases of peritonitis, either of itself, or as a participator in mixed infections. However, in a large proportion of the cases examined, cocci (especially streptococci) were a prominent feature.

I would next divert to make a few remarks upon the method of examination. In the first place I would insist upon the importance of the examination of the *omentum*.

It has been shown¹ that substances (bacilli, Indian ink, &c.) become deposited upon the omentum after introduction into the peritoneal cavity; so much so that the peritoneal fluid may be quite sterile, microscopically and culturally, whilst upon the omentum (and other peritoneal membranes) numerous, even infinite numbers of organisms are to be found. It therefore follows that mere failure to find microbes in peritoneal fluid is not sufficient evidence to stamp a case as "chemical peritonitis" (Tavel and Lanz).² No doubt in such cases the prime cause is toxic or chemical, yet if living infective agents are still present the inference clearly is that they may have had some share in the production of the fatal dose. But a mere examination of the local condition in the peritoneal cavity is not sufficient to give conclusive evidence as to the actual pathogenic microbe which has been the cause of death.

The two places in this connection are—1. The lymph paths and glands concerned with the peritoneum. 2. The blood.

It is curious that the lymph drains of the largest lymph space in the body are not even hinted at in works on anatomy. I have pointed out elsewhere (*loc. cit.*) that the most important of the lymph channels which drain the peritoneal cavity are those which pass up along the anterior mediastinum. This is founded on the experimental evidence of the passage of bacilli and coloured materials along these tracts, and is confirmed by the dissection of a case of ruptured tubal gestation in which the lymph paths were beautifully injected with blood; as well as of observations on tuberculous and acute peritonitis, in both of which these lymphatic glands are found to be affected.

In cases of acute peritonitis the glands of the anterior mediastinum (those in the first intercostal space are the most constant and convenient to examine) are reddened,

¹ Brit. Assoc., 1896, and Gruber and Durham, 'Wiener klin. Wochenschr.', 1896.

² The same holds good for examinations of hernial sacs and fluid, which have often been said to be sterile (*vide* Brentano).

swollen, and are found to contain microbes; sometimes they are necrotic, sometimes they are more or less hæmorrhagic. These being paths whereby microbes pass from the peritoneal cavity to the blood to cause bacteriæmia (Kocher) or general infection, it is obviously necessary to examine them.

The examination of the blood has been made by sucking some from the jugular vein with sterile pipettes. Careful antisepsis and asepsis have been employed to prevent contamination; and many control cultures have been made from non-septic cases in order to test the methods employed.

A complete examination of a case of peritonitis entails, therefore, the following investigation:—Microscopical (hanging drop and stained specimens) and cultural from peritoneal fluid, omentum, anterior mediastinal gland juice, blood, as well as examinations of the pseudo-membranous deposits and flakes ("lymph," "fibrin") which are often present, and are cellular (not fibrinous) in structure. (See Table, pp. 203, 204.)

The Cases 8, 9, 14, 17, and 19 are especially interesting in the light of A. Fränkel's investigations. This author shows that many cases which die shortly after operation, and whose deaths are ascribed to shock, are in reality cases of acute septic infection.

Observations upon animals (which may die as early as four or five hours after infection) show that the more virulent the infection the less marked are the local signs of peritonitis. In the post-mortem room, unless a quantity of pus is found in the peritoneum, the case is often put down as "no peritonitis:" observation of the omentum and the anterior mediastinal lymph tracts, combined with bacteriological examination, will set this matter right. It is only when animals die of relatively subacute peritoneal infection that pus, flakes of cells, pseudo-membranes, and the like are to be found.

[The whole question of death in comatose states, shock collapse, &c., requires further investigation. At present I have only examined one case of "uræmic coma;" in this

the blood and organs were literally swarming with *Staphylococcus aureus*. In this case the examination was made within three hours of death.]

These cases are sufficient to show that the bacteria in peritonitis are of various kinds, and that it is not possible to be certain what those kinds will be in given cases. However, it will be seen that cocci, especially streptococci, must be regarded as important infecting agents even in poly-infections of the peritoneum.

These cases, with those of the authors quoted above (Tavel and Lanz, Predöhl, &c.), and Dr. Klein's and Vincent's observations on perforation in typhoid fever (see Case 13), all point towards the advisability of the prophylactic use of anti-streptococcal serum the day before an operation is undertaken in dangerous conditions.

It will be borne in mind that the peritoneum will be strongly protected specifically against streptococcal invasion; whilst at the same time it will have an increased resisting power towards other kinds of pathogenic microbes.

It has already been shown that the varieties of *B. coli* obtained from cases of peritonitis do not all react mutually with the sera obtained by their means (Durham, loc. cit.); there is therefore no advantage in using one kind of *B. coli* serum for prophylactic injection.

Of indifferent substances nuclein or nucleic acid seems to be the most active; but as far as can be seen at present the anti-streptococcal serum promises better results.

The second point towards which I propose to call your attention is in regard to the treatment of general suppurative peritonitis. The only treatment in which the hope of saving the patient lies consists in the cleansing of the peritoneal cavity, combined with the sealing up of the source of infection if any, and possibly also the administration of a proper serum therapeutically.

It is to the matter of the technique of the flushing or washing process that I venture to direct your attention.

I have already adverted to the fact that the peritoneal

cavity rids itself of small foreign bodies (bacteria, &c.) partly by determining deposit on the omentum, &c., and partly by the intervention of the lymph flow. Also that the peritoneal fluid may contain but few microbes, or indeed be quite sterile, at a time when the omentum shows abundant hosts of them.

This was the case in the rabbit No. 2, referred to at the commencement of the paper, as well as in a number of other animals during the course of my experiments.

It appears that the peristaltic movements of the intestines are largely responsible for the deposition upon the omentum, whereby in small animals the omentum becomes rolled up. Owing to this function of the omentum being dependent upon the intestinal movements, we can readily understand the *rationale* of the otherwise empirical use of cathartics suggested by Lawson Tait; by such means the peristalsis is established, and improved circulation is determined to the mesenterial vessels, from which the chief transudation of fluids and leucocytes takes place.

In the laboratory and in the post-mortem room it is found that the majority of organisms and exudation cells are found about the omentum, the surfaces of the liver, spleen, and diaphragm,—in fact, the upper parts of the peritoneum. It seems clear, therefore, that in cleansing the abdominal cavity *especial attention should be paid to thoroughly washing and mopping these parts—the omentum, and between the liver and diaphragm*,—not that the other regions should be neglected.

In conclusion it may be said that (1) the prophylactic intra-peritoneal injection of suitable sterile material (such as anti-streptococcal serum, sterile broth, or some blend of different kinds of serum) twenty-four hours before proceeding to operations on cases where the peritoneum is in danger of being contaminated by the contents of abscesses, &c., and (2) the particular cleansing of the omentum and upper part of the peritoneal cavity, in operating upon cases of general peritonitis, both appear to be rational, and practical from being based upon experimental observations.

Finally I must express my thanks to the physicians and surgeons of Guy's Hospital for kindly allowing me to use the cases which were under their care, and express the hope that many otherwise hopeless cases may be saved by the precautions and technique which have been indicated.

The following literature may be consulted in connection with the above remarks :

BARBACCI.—“Ueber die Aetiologie und Pathogenese der Peritonitis durch Perforation,” ‘Centralbl. f. allg. Path.,’ iv, 1893, p. 769.

BRENTANO.—“Die Ergebnisse bacteriologischer Bruchwasseruntersuchungen,” ‘Deut. Zeit. f. Chirurg.,’ xliii, 1896, p. 287.

BUMM.—“Zur Aetiologie der sept. Peritonitis,” ‘München. med. Wochenschrift,’ 1890, p. 185.

CURNOW.—“Lectures on Lymphatics,” ‘Lancet,’ ii, 1879.

E. FRÄNKEL.—‘München. med. Wochenschr.,’ 1890, pp. 23 and 202.

A. FRÄNKEL.—“Ueber peritoneal Infection,” ‘Wiener klin. Wochenschr.,’ 1891.

M. HAHN.—“Zur Leichendiagnose der pyaemischen und septikaemischen Processen,” ‘Virchow's Archiv,’ cxxiii, 1891, p. 1.

M. HAHN.—“Ueber die Steigerung der natürlichen Widerstandsfähigkeit durch Erzeugung von Hyperleucocytose,” ‘Berlin. klin. Wochenschr.,’ 1896, p. 864.

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VINCENT.—“Étude sur les Résultats de l’Association du Streptocoque et du Bacille Typhique,” ‘Ann. Inst. Pasteur,’ vii, 1893, p. 141.

WURTZ AND HERMANN.—“De la Présence fréquente du *B. coli* dans les Cadavres,” ‘Arch. Méd. Exp.,’ 1891, p. 734.

Postscript.—Dr. Muscatello, of Turin, has sent me a copy of his paper on “Absorption from the Peritoneal Cavity” (‘Virchow’s Archiv,’ cxlii, 1895). In this he calls attention to the importance of the lymphatic tracts of the anterior mediastinum; and I must offer an apology for having overlooked his valuable contribution.—*April, 1897.*

(For report of the discussion on this paper, see ‘Proceedings of the Royal Medical and Chirurgical Society,’ Third Series, vol. ix, p. 91.)

No.	Sex. Age.	Diagnosis, &c.	Since Death.	Bacteriological Examination, &c.
1	F. 13½	Double suppurative salpingitis; suppurative peritonitis; celiotomy; lavage; drainage. Death after 9 days	14 hours	Bacillus coli; streptococci; blood sterile to broth culture.
2	F. 9	Perityphlitic abscess; operation. Death 4th day; suppurative peritonitis	19 hours	Bacillus coli; streptococci.
3	M. 15	Perityphlitis; perforated appendix; general suppurative peritonitis; operation not undertaken	3 hours	Streptococci very abundant; a few large non-liquefying bacilli; only streptococci from anterior mediastinal gland.
4	M. 7	Perforated appendix; suppurative peritonitis; no operation	3½ hours	Abundant small bacillus unidentified; few B. coli; few cocci.
5	M. 42	Recurrent attacks; ulcerative colitis; suppurative peritonitis; no operation	19 hours	Poly-infection; streptococci very abundant; bacillus growing in long threads, abundant; some B. coli.
6	F. 6½	Strangulated femoral hernia; bowel gangrenous; primary resection; Murphy's button. Death 4th day; suppurative peritonitis	47 hours	Poly-infection locally; several kinds of bacilli, also streptococci; ant. med. gland pure culture streptococci; blood sterile to culture.
7	F. 42	Strangulated umbilical hernia; resection, Murphy's button. Death 10 hours after operation = 33 hours after strangulation	24 hours	All organs examined with large, anaërobic, non-motile bacillus, also small bacillus which did not grow; ant. med. gland very red, swollen, and soft.
8	F. 41	Femoral strangulated hernia; perforation; resection, Murphy's button; acute peritonitis. Death within 24 hours	4 hours	Peritoneal fluid sterile; omentum and anterior med. glands abundant Staphylococcus aureus; blood sterile.
9	M. 10	Intussusception; celiotomy; "collapse;" death. Several operations, death 15 hours after last; acute peritonitis	9 hours	Abundant B. coli and staphylococci.
10	M. 2½	Intussusception; celiotomy; Murphy's button. Death	24 hours	B. coli and streptococci; droplet of juice of ant. med. gland remained sterile.

No.	Sex.	Age.	Diagnosis, &c.	Since Death.	Bacteriological Examination, &c.
11	M.	64	Epithelioma of caecum; obstruction; peritonitis; n. operation. Death	25 hours	<i>B. fluorescens non-liquefaciens</i> (much like <i>B. coli</i> for 4 or 5 days) and staphylococci.
12	M.	56	Epithelioma recti; inguinal colotomy; suppurative peritonitis. Death	17 hours	Locally abundant streptococci and small bacillus; anterior med. gland only cocci.
13	M.	24	Enterica; perforation. Death.	8½ hours	Locally poly-infection; especially abundant streptococci; also <i>B. coli</i> and other bacilli.
14	F.	58	Epithelioma pharyngis; gastrostomy; "exhaustion." Death 40 hours after operation	15 hours	Very abundant streptococci; also <i>B. coli</i> .
15	F.	23	Gonorrhoeal pyosalpinx; celiotomy; suppurative peritonitis	31 hours	Cocci and bacillus resembling <i>B. coli</i> , but which slowly liquefied gelatine; ant. med. glands only cocci, which did not grow.
16	F.	63	Twisted ovarian tumour; celiotomy; "diabetic coma." Death 4th day; limited examination	12 hours	Microscopically cocci; only gelatine cultures made, which did not grow.
17	F.	41	Fibroids; celiotomy; 4th day intestinal obstruction due to kinking; operation. Death after 3 hours	11½ hours	Locally ant. med. gland and blood yielded pure growth of <i>Staphylococcus aureus</i> ; also abundant microscopically.
18	F.	22	Abscess around pyosalpinx; rupture; suppurative peritonitis	15 hours	Locally poly-infection; <i>B. coli</i> and cocci; blood and ant. med. gland pure <i>Staphylococcus aureus</i> .
19	F.	28	Suppurating ovarian cyst; celiotomy; escape of pus into peritoneum. Death 30 hours post operationem	12 hours	Locally, and ant. med. gland <i>Staphylococcus albus</i> .
20	F.	48	Sloughing fibroids; pelvic abscess; celiotomy. Death 3rd day; acute peritonitis	3 hours	Locally streptococci and staphylococci; blood pure <i>Staphylococcus citreus</i> .

A CASE
OF
LARGE SOLID TUMOUR

REMOVED WITH SUCCESS FROM THE RETRO-PERITONEAL
SPACE

BY
A. MARMADUKE SHEILD, M.B., F.R.C.S.

Received October 26th, 1896—Read March 9th, 1897

A WIDOW aged 50 was admitted into St. George's Hospital under my care on September 15th, 1896, being recommended to me for treatment by Dr. Gould May. Menstruation had ceased twelve months ago. She had had two children and no miscarriages. The uterus had been completely prolapsed for twelve months, and before then the woman had difficulty with micturition, the prolapse gradually getting worse. For four months she had noticed a large abdominal swelling, which had slowly increased. On examination a tumour of considerable size fills the abdominal cavity, being more pronounced in outline towards the left flank, and reaching above nearly to the ensiform cartilage. There is a well-marked area of resonance round it, and it is quite dull in front. Below, it seems to pass into the pelvis. The growth feels

elastic and semi-fluctuating, but there is no distinct thrill or percussion wave. No doubt was felt that it was fluid in consistence, and I even had suspicions it might be an excessively distended bladder. The uterus was completely prolapsed, and when it was reduced, with some difficulty a catheter could be passed into the bladder, evacuating that organ, without any effect upon the size of the abdominal tumour. The opinions expressed were in favour of the growth being ovarian in origin.

On September 22nd the operation was performed. In making the usual incision it was at once noted that something unusual was going to present, in the number of fibrous laminæ containing large and branching veins, which needed division, and the ordinary peritoneal investment was not recognised. After dividing many layers, the surface of the tumour, white and glistening exactly like fat, was exposed. It was found universally adherent to its capsule. An incision was made into the growth, under the idea it was cystic. It was then found for the first time that the tumour was a soft solid, and very deeply and firmly seated. Free hæmorrhage was taking place from the incision, and seeing the hopeless prospects for the patient if the wound was closed and the operation abandoned, I determined to remove the growth at all hazards. As the tumour did not present a malignant aspect, but rather resembled a lipoma, I at once prolonged the incision upwards nearly to the ensiform cartilage, and here some coils of intestine, including the transverse colon, were lying quite flattened in front of the tumour and adherent to its thick capsule. The arrangement of parts was very confused; there were numerous laminæ of thick membranes, and I was careful to work absolutely upon the surface of the tumour. This was effected by careful peeling away of the deepest fibrous laminæ. On either side the same difficulty was experienced, some intestine and apparently flattened-out mesentery lying in front of the tumour. At length I was able to get my hand

behind the fundus of the growth and raise it from the spine. It separated readily from the aorta and vena cava. Moreover some lobes and lobules became apparent, and these shelled out with ease from the tissue on either side of the spine, giving me the assurance that the tumour was innocent in nature, and affording great encouragement to proceed.

The mass was now delivered and supported by an assistant, and separation of the bladder was proceeded with. A free transverse incision was made with a scalpel across the front of the tumour, and the bladder readily peeled away with a lamina of tumour capsule adherent behind it. A number of dense adhesions, containing very large vessels, connected the growth with the tissue about the front of the lumbar spine and brim of the pelvis. These were secured by transfixing and careful tying with strong silk. The condition of the patient now became serious, and six or eight thick adhesions were rapidly secured and divided, and the tumour removed. Hot water flushing at once restored the pulse and colour of the patient. No loss of time was allowed to intervene before the wound was closed, a tube being introduced below. The operation lasted forty minutes, and its later stages were performed with all possible rapidity.

The tumour in the fresh state was exceedingly soft and elastic, white and wax-like in colour. It contained a great amount of serum, which drained from it in quantities. After soaking a few days in spirits it weighed nearly nine pounds, and shrank considerably; in the fresh state it must have weighed much more. My colleague, Dr. Rolleston, pronounced the growth to be a soft fibroma, containing unstriped muscle, so that the structure resembled that of a uterine fibro-myoma, but very soft and œdematous.

The patient suffered from severe collapse after the operation, and was only kept alive by careful rectal feeding and small quantities of champagne by the mouth.

Several ounces of blood-stained fluid were drawn through the tube, which was removed on the second day. On the fourth day, as the abdomen was distended, the bowels were moved by salines and assafoetida enemata. After the bowels acted her case ceased to give any anxiety. A small sero-sanguineous collection formed at the lower part of the wound on the fourteenth day; this was evacuated and washed out, and the sinus soon closed. The sutures were removed, and the abdominal wound proved soundly healed by the end of the third week. Great care and attention was devoted to the after treatment of this case by Mr. Blackett, then house surgeon, and I wish to acknowledge his valuable services in this respect.

The complete prolapse of the uterus in this case put out of consideration the probability of a large abdominal tumour being of a uterine origin, and I was much surprised at the report of the microscopical examination. The growth may originally have been a uterine fibroid, which had become attached superiorly, and detached from the uterus as the latter descended out of the pelvis. A striking feature was the extraordinary number of adventitious adhesions. The tumour seemed to receive blood-supply from every membrane it lay in contact with. The anterior and lateral adhesions were readily broken down by firm sweeps with the fingers, but sometimes they bled and oozed alarmingly. It is, of course, possible that this tumour, though a fibro-myoma, may have originated apart from the uterus. Such a question is most difficult to settle, but tissue containing unstriped muscle is known to exist in the broad ligaments about the brim of the pelvis behind the peritoneum as high as the renal capsule. The comparatively rapid growth is in favour of this view.

The mesentery seemed to have been entirely displaced by pressure behind. Traces of it were plainly to be seen in front of the tumour, and some of the many vascular adhesions separated behind may have contained mesenteric vessels, but the parts were too confused to

admit of certainty, and the patient too ill to allow of prolonged investigation.

The exact position of the tumour in the present instance is most difficult to define, for the anatomical distribution of the parts was so confused, that had I waited to be clear where I was working, the tumour would probably have never been removed. The presence of flattened-out intestine and mesentery in front of this growth, and the many membranous structures which had to be divided before the real tumour substance was exposed, made me believe that the main bulk of the growth at any rate was behind the peritoneum. The aorta and vena cava were also plainly to be felt on shelling the mass away posteriorly. I am not at all ready to explain how this remarkable growth reached the position it occupied if primarily uterine, but I only state the facts undoubtedly observed.

The adhesion of the transverse colon and some coils of small intestine to the front of the tumour was very close. Indeed, the colon might readily have been cut across unless the tissues in enlarging the wound had been cautiously divided. The main proceeding which enabled this operation to be completed without disaster and in a reasonably short space of time was undoubtedly the length of the incision. I feel sure that in complicated abdominal cases of this nature it is absolutely essential to see what one is doing if the operation is to be performed with that celerity which is essential to ultimate success. Prolonged groping and tearing through an insufficient opening is a fertile source of delay and disaster; and it is most essential to proportion the length of the incision to the difficulties and bulk of the tumour.

Another important operative consideration to which I would draw attention is the importance of working along the surface of the tumour itself. The laminæ overlying the growth in this case were so numerous that I constantly found myself separating membrane from membrane, rather

than membrane from tumour. When this was the case I incised with the scalpel until tumour substance was seen. In this way the bladder separated anteriorly with remarkable ease, though it seemed at first quite incorporated with the tumour.

The literature and recorded cases of retro-peritoneal tumours is not very large, and must serve as an apology for the brevity of the illustrative remarks upon this case. Leaving aside purulent collections, extravasations of blood, and cysts, as hydatids, dermoids, or pancreatic cysts, the solid tumours in this region resolve themselves into innocent growths, as the fibro-fatty and more rarely the fibro-myomata, and sarcomatous growths, simple or mixed with cartilage. I have seen two cases where cartilaginous tumours of huge size originated from the osseous structures of the spine and ribs; but these in no sense originated in the retro-peritoneal tissue, and should not be classified with the affections of this region. The fibro-lipomata seem to be the more common, and these tumours may attain to a large size. Their situation and relations are very complicated, and they may be so soft as to exactly simulate collections of fluid. All this is well seen in Cooper Foster's celebrated case, where the tumour weighed fifty-five pounds, and was tapped in mistake for ascites. After the decease of the patient this great tumour was found to consist of pure fat, and the relations of the colon and small intestines to the front of the growth were just as is related in the case reported to-night ('Path. Soc. Trans.,' vol. xix, p. 246).

Wells, in his work on abdominal tumours, speaks of three tumours apparently situated behind the peritoneum. The first, weighing twenty pounds, was a fibro-lipoma, and this patient died. The second was malignant, and the operation had to be abandoned. The third, removed from a lady in Pomerania, was called by Virchow a "fibroma molluscum cysticum," but its true nature seems a little doubtful ('Abdominal Tumours,' p. 24).

Certain tumours of the kidney and renal capsule have

contained unstriped muscle (Morris's 'Diseases of the Kidney,' p. 499), but tumours such as I have related this evening seem exceedingly rare. The subject is not alluded to in the more modern books on tumours and abdominal surgery.

Mr. William Anderson, in his excellent opening of the discussion on retro-peritoneal affections at the meeting of the British Medical Association at Carlisle, refers at length to the tumours of the retro-peritoneal space ('Brit. Med. Journ.,' Oct. 17th, 1896). It is obvious from his researches that the most common are lipomata, fibro-lipomata, and sarcomata. He mentions that fibromyomata may originate in the scattered fibrous bands about the root of the mesentery and broad ligament. He refers to one case at the Tokyo Hospital, where a girl of nine had a number of tumours of a fibro-myomatous nature removed from behind the peritoneum. This patient died of shock. He also alludes to a case where Péan removed a similar tumour from the root of the mesentery which weighed eighteen pounds.

Beyond the two cases referred to by Mr. Anderson, I can find none reported in this country which resemble the case related. Lipomata or fibro-lipomata of huge size have been recorded, among others by Pick ('Path. Soc. Trans.,' 1869, p. 337), Meredith ('Clin. Soc. Trans.,' 1887), and Treves ('Clin. Soc. Trans.,' 1893, p. 101). Mr. Anderson refers to other cases under the care of Continental surgeons. In all the cases where removal was attempted of retro-peritoneal fatty tumours, the operations were very difficult on account of the complicated adhesions and the nature of distribution of the viscera anteriorly. Cases of retro-peritoneal sarcomata have been brought forward in this country by Thornton ('Brit. Med. Journ.,' 1882, vol. ii) and by Lockwood ('Med. Soc. Trans.,' 1895, p. 1).

The removal of malignant growths from this situation must always be peculiarly hazardous, especially if they are soft and vascular; and probably in the majority of

instances the surgeon will do wisely, when the nature of the tumour is verified by exploration, not to attempt the operation. It is worth remembering that retro-peritoneal sarcomata may be secondary to growths elsewhere. I have this year seen a remarkable instance of this nature, when a large growth, easily to be felt anteriorly and posteriorly in the loin, originated by lymphatic infection from a nodule of sarcoma in the testis. No post-mortem examination was held, but the diagnosis was very clear, and was verified by Sir William Broadbent. A similar case to this is mentioned by Sir Hugh Beever, where a large tumour having the colon in front of it formed after removal of the left testis ('Med. Soc. Trans.,' vol. xvii, p. 329).

Mr. Lockwood fully alludes to the difficulties of diagnosing sarcomatous tumours in this region ('Med. Soc. Trans.,' vol. xvii). In his two cases the colon and intestines lay in front of the tumour, as is usually observed. He alludes to the possibility of getting areas of resonance in front of these growths. In the early stages this would, without doubt, be possible. But when the tumours grow to a huge size the gut gets so flattened that this important symptom may be quite absent. It is very essential that retro-peritoneal tumours should be fully reported as they occur. Thus only shall we in time recognise their diagnostic features and appropriate treatment; and it is largely with this view I have ventured to bring this case forward, for it may add a small though important item to a class of affections regarding which little really is known.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 90.)

THE PARASITE OF MALARIA IN THE FEVERS OF SIERRA LEONE

BY

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ARMY MEDICAL STAFF

(COMMUNICATED BY DR. GEORGE THIN)

Received January 13th—Read March 23rd, 1897

THE well-known virulence of the malarial fevers which prevail on the west coast of Africa continues until the present time with unabated force. Europeans and natives are alike subject to their ravages, although they exact a higher tribute from the former. When not fatal such fevers often render life a burden for many years, whilst a few fortunate individuals seem, at least for a long time, to be proof against their attacks.

Sierra Leone (where the following observations were made) is a highly mountainous district, both its valleys and mountains being covered with a very luxuriant vegetation. The soil consists of a porous red earth, which appears to play an important part in the causation of malaria.

The river Roquette, which has a large volume of water, opens into the sea at the native town of Freetown, half a

mile inland from which are stationed the principal barracks, which are built on a hill about 600 feet high. Mangrove swamps exist near Freetown, but are covered by the tide. The average daily temperature is about 80° F., and the annual rainfall about 150 inches. The rainy season lasts from May till October. There is no cold season. The heat is trying from the large amount of moisture present in the atmosphere.

My experience in this disease was gained during a residence of two years at Sierra Leone, where I was occupied in attending the officers and men of Her Majesty's West India Regiment. The two years were not consecutive, being separated by the usual interval of one year. As I made it my practice, during both periods of residence, to examine the blood of all the fever patients with whom I came in contact, I am within the mark when I state that I must have examined the blood of at least 400 different patients suffering from fever. I have taken notes of the observations in 50 cases. The cases which I shall relate in this paper are selected from the 50, and are fair average examples of all the others. During part of the time I used a Swift's $\frac{1}{12}$ in. oil immersion, and for the rest of the time a Zeiss $\frac{1}{12}$. Both these objectives showed the parasite in the fresh blood sufficiently well. Examination of the blood was discontinued after the patients left the hospital. In many cases they had suffered from fever for some time before admission. They were detained in hospital until free from fever. The average duration of the stay in hospital of the fever cases in Sierra Leone which I observed was about a week. In most cases that short period was sufficient to cure the fever for the time being. In many instances the fever symptoms disappeared within a day, sometimes to reappear a few days afterwards; but sometimes the temperature did not again rise.

The first attack of malarial fever experienced by newcomers is usually of the quotidian remittent type. The onset is often sudden, and is frequently unattended by rigors.

The symptoms in an ordinary attack are as follows. The patient complains of headache, and pains in the trunk and limbs, and occasionally of severe giddiness. The tongue is furred, and there is frequently nausea with very distressing bilious vomiting. Constipation is common, but in some cases I have seen severe diarrhoea. Occasionally there is no disturbance of the bowels whatever. A dull aching, and sometimes an acute pain is present in the hepatic region. Jaundice is rare. The spleen is not enlarged to an appreciable extent. The temperature on the first day ranges between 103° F. and 105° F.; on the second morning there is usually a remission of two to three degrees, and towards evening a rise to 105° F.; on the third morning there is again a remission of several degrees, and in the evening another exacerbation; on the fourth morning the temperature is often normal, and in the evening there may be again a slight exacerbation— 100° F. to 101° F.; on the fifth morning the temperature is normal, and as a rule remains so.

Quinine is so generally given in these cases that the above account of the course of the symptoms necessarily refers to cases that have been treated by the drug, but there is reason to believe that even without quinine the course of the fever would be somewhat similar. When I myself suffered from the fever, the quinine which I took was at once rejected by vomiting to such an extent that I do not think that any of it was retained; certainly none for the first three or four days, notwithstanding which I had a remission each morning.

The skin, which is at first hot and dry, soon becomes covered with a profuse perspiration, although for some days there is no corresponding fall of temperature.

Delirium, if present, is generally slight, but for several nights there are most distressing dreams, and a tendency to magnify the most trifling occurrences. The symptoms are, in many respects, similar to those which are characteristic of delirium tremens.

The pulse and respiration rates are directly propor-

tional to the temperature. The urine frequently contains bile.

About the fourth day of the fever the tongue begins to clean, and there is a desire for solid food. The other evidences of fever disappear, and in a week the patient feels quite well.

Subsequent attacks occur at irregular intervals, and often last only a few hours. In many of them the three stages of an ague fit are present. In the men of the West India Regiment, recruited in the West Indies, and consisting of negroes and a large proportion of mulattos, malarial fever, except in some bad cases, does not continue for many hours, but the admissions to hospital are frequent, and there are usually several attacks during each admission.

Pyrexia, in a few cases, lasts for weeks, the temperature being about 101° F., and often remitting a degree in the morning. One such case ended fatally in the fourth week, although the patient was invalided to Grand Canary in the second week of the disease. This result was chiefly due to the debilitating effects of a long arduous expedition after two years' residence in Sierra Leone.

Dr. Prout ('Lancet,' 1891, vol. ii, p. 226) gives an account of the malarial fevers observed on the Gold Coast. The symptoms which he describes in these fevers are essentially the same as those which I have given as generally attending the fevers at Sierra Leone. It will be noticed that I have not laid the same stress on enlargement and tenderness of the spleen which Dr. Prout has done; although in chronic cases enlargement of the spleen is common, in many acute cases which I observed of a short duration I did not notice much tenderness or enlargement of that organ.

Fatal cases.—In the fatal cases I have seen, death with one exception was preceded by several hours or days of unconsciousness. In one case there were hysterical fits for five hours before death, the patient struggling violently with his attendants, whistling, singing, shouting, and

breathing about sixty per minute ; these fits were preceded by two days' delirium, and at no time during this period was there any return of consciousness.

The temperature varies considerably ; in some it runs up to 107° or 108° F. According to my experience this is most likely to occur in new-comers ; but in those who have undergone considerable hardship in the colony, or have been resident a long time, the temperature shortly before death may be normal, or not more than two or three degrees above normal.

Death occurred in one case which I observed from hæmorrhage from the bowels. The hæmorrhage occurred suddenly, and continued till he died, about nine hours later. In several cases in which the fever was pursuing a mild course serious symptoms set in suddenly and unexpectedly, and the case terminated fatally after a shorter or longer period. During the earlier and milder stages of these attacks there was no symptom which led me to expect a fatal result.

In examining the blood, fresh and dried preparations were made in the usual way. Specimens were stained with eosin and methylene blue, or Ehrlich's hæmatoxylin, and in some cases a drop of very dilute solution of methylene blue was added to fresh blood. Parasites were always found during the whole of the attack and for several days after.

My examinations were made from 10 a.m. till 2 p.m., and again from 4.30 p.m. to 6.30 p.m. As parasites were found in every case at these times whilst the fever lasted, it is presumable that they would have been found had the examinations been made at other times of the day, and the inference may be drawn that parasites in these fevers can be found in blood taken from the finger at any time during the course of the disease.

With the exception of an account of the histology of the tissues in a fatal case of pernicious malaria at Sierra Leone by Dr. Thin, which is published in vol. lxxix of the 'Medico-Chirurgical Transactions,' nothing appears to

have been published regarding the parasite of malaria which occurs in that part of the west coast.

I have only succeeded in distinguishing one kind of parasite, which was, in a proportion of cases, accompanied or followed by the so-called crescent forms. I exclude a case of tertian fever to be subsequently referred to. In the unpigmented stage this parasite appears as an extremely small bright speck on the surface of the red corpuscle, from which it cannot be detached by any ordinary pressure. At first it is constantly changing its shape, at one time spherical, and at another throwing out processes which, remaining visible for a number of seconds, are gradually withdrawn, and an irregular shape is maintained for some time. Instead of this amoeboid motion the parasite every now and then suddenly contracts to half its size, and just as quickly resumes its original condition. This movement is repeated a number of times. Finally, after several hours the parasite assumes a spherical or slightly irregular form, in the centre of which what appears to be a small portion of the red cell is visible. The central portion stains with methylene blue in the youngest parasites. At a slightly more advanced stage only the periphery of the parasite takes on a blue stain, and one or two deeply stained nucleoli can be easily made out. A more advanced stage is evident in a few cases where the whole parasite containing a mass of pigment stains blue. In most cases there is only one parasite attached to a red blood-cell. The largest number I have seen occupying one red cell is five, all of which were at the same stage of development.

In some specimens stained with eosin and methylene blue I have observed two somewhat conical-shaped parasites united by their bases, each parasite having a nucleolus and with only the periphery stained blue.

I often observed the same parasites for hours continuously, and although the movements continued active the development of the parasite never progressed, as it presumably would have done if the blood had not been removed from the body.

The affected red cells did not seem to be altered as regards colour, size, and shape.

I never saw the so-called brassy-red corpuscle (the "*ottonati*" of the Italian writers), although I looked carefully for it.

In the early stage of pigmentation the parasite has enlarged slightly, and contains several minute grains of pigment. The sporulating stage was never observed by me in the peripheral blood. It is best seen, according to my observations, in the fresh blood removed from a small vessel on the surface of the brain soon after death. The effect of the parasite on the movement of the affected cells is very evident in such a case, for while the normal red cells were influenced by currents in the field of the microscope, those containing the parasite were perfectly motionless. It was then seen that sporulation occurs while the parasite does not occupy more than a third of the red cell. The spores became indistinct so soon after the death of the patient that I found it difficult to count them, but in one case in which I succeeded in doing so I saw five spores surrounding a small central block of pigment.

Crescents were found in varying numbers in most of the cases. In length they varied from about 8 to 12 mm., and in breadth from 3 to 5 mm. In most cases the pigment is massed near the centre of the crescent. In the larger crescents the pigment appears to lie in a differentiated structure in the centre, and in one crescent I saw the pigment lying beside this structure. The crescent is often seen in close relation to several blood-corpuscles, from which it can be readily detached by pressure. It does not appear to be capable of independent movement.

I have on several occasions seen an active movement in the central pigment of the crescent body, the minute spheres and rods of which it is composed moving rapidly from one clear space to another in the central portion of the parasite. This movement sometimes continued for several hours. Mannaberg states that the concentrated

pigment is invariably quiescent (New Sydenham Society translation, p. 284).

I have never observed the crescent change into a spherical form with the formation of flagella. In the light of the information contained in the letter to the 'Lancet' by Dr. Marshall (October 24th, 1896) this is probably owing to the moist warm atmosphere in which the specimens were examined, which would tend to keep the blood-plasma unaltered for a considerable length of time. I never saw flagella, although the crescents were often under observation for many hours. A number of the spherical forms still retained the outline of the red cell in which they had developed, as was signified by the delicate envelope of red blood-cell substance which surrounded them. The contained pigment was sometimes in the form of comparatively large spheres, several of which became detached from the others, and moved round the interior just within the outline of the red cell.

Dr. Prout, in the paper already referred to, gives the results of microscopical observations made by him in ten cases. Examination of the blood was made in ten cases, partly European, partly native, including two cases of bilious remittent, one of hæmoglobinuria, the rest mild intermittent. Only fresh unstained specimens were examined, taken generally when the temperature was rising, and on one or two occasions at different intervals during the attack. In eight cases there were distinct changes in the red cells, divided by Dr. Prout into five kinds:—1st. Brightly refracting rod-like bodies occurred in three cases, and in one of them were very numerous. They varied in number and size, possessed a certain movement described as pulsatile, and occasionally a slight alteration of their position in the corpuscle occurred. 2nd. Brightly refracting round spots of different sizes, sometimes combined with the rod forms. 3rd. Large circular bodies like vacuoles, in the centre, or to the side of the corpuscle, sometimes with rods. 4th. Irregular bodies which may be regarded as transition stages between the above forms. 5th. In three cases

bodies like a tadpole or spermatozoon with an oval head, and tapering filament attached. Like the other bodies they were translucent and possessed limited movement. They are not so common as the others, four being the largest number in one preparation. None of these bodies contained pigment. Pigmented bodies were seen in five cases. 1st. Small corpuscles about the size of a leucocyte with dark brown pigment granules distributed evenly throughout the cell. 2nd. Bodies twice to thrice the size of a leucocyte containing similar granules of pigment, but arranged round clear spaces. 3rd. Pigmented bodies showing amœboid movement. 4th. Amœboid bodies containing large masses of pigment. These differ from the bodies described above in the character of the pigment, which is collected in large masses instead of fine rounded granules, and are probably the phagocytes on whose scavenging properties Carter lays so much stress. It is possible that all these forms are merely stages of the same body.

Dr. Prout did not detect pigmented crescents, spheres, or flagellated organisms. The intra-corpuscular forms were present before the paroxysm, and while the temperature was rising, and usually disappeared under treatment; while the pigmented forms were found at all stages, but persisted for a considerable time after the attack had ceased. In one case the whole of the bodies were found; in one there were rods, clear spots, and pigmented bodies; in one, rods, vacuoles, and pigmented bodies; in two, vacuoles and tadpole bodies; in three, vacuoles only; and in two, pigmented bodies only.

I am unable to identify most of the appearances described by Dr. Prout with the appearances which I consider to be characteristic of the parasites observed by me at Sierra Leone. I miss in his description the ring-shaped parasite which I found so universal in my cases. The larger pigmented bodies described by him, and shown in his drawings, will be generally, I believe, considered to be pigment-bearing leucocytes. As so little has yet been

written on the parasite of West African fevers, I have considered it advisable to give this *résumé* of the observations recorded by Dr. Prout, so that those who are specially interested in the subject may have an opportunity of referring to his original paper.

The following eleven cases are selected so as to illustrate the usual course of the fever in its relation to the parasite. In all the observations the blood was taken from the finger. I never saw any unpigmented forms free in the blood.

CASE 1.—Private B—, admitted to hospital on January 23rd, 1896, complaining of having suffered from fever since the 20th. Temperature on admission 103° F.

The blood examined at noon contained a number of free crescents. There was no movement in the crescents or their pigment. There was about a dozen in one specimen. Many of the crescents were elongated and narrow, nearly twice the size of a red cell. There were also minute unpigmented parasites in the red cells, similar to those seen in other specimens. There was a good deal of free pigment in the blood. At 5 p.m. temp. 101° F. Crescents still present in the blood. The patient received 5 grains of calomel, and later 5 grains of antifebrin, and two 15-grain doses of quinine.

January 24th.—Morning temperature normal. Patient feels better. Blood still contained crescents, but they are less numerous than on the previous day. One oval young form which was tinged externally with hæmoglobin. No evidence of spore formation. Movements of pigment visible in the crescents. Numerous pigmented leucocytes and a good deal of free pigment. Evening temp. 99° F.

25th.—Morning temperature normal. Patient feels quite well. Examined specimens of blood, one taken yesterday evening and one this morning. Crescents present in both, probably more numerous in the former. One crescent contained two separate bodies, one deeply pigmented, and the other having one or two minute dots.

At one time they appeared to unite, but in the end no special change occurred. There were a few unpigmented parasites and numerous pigmented leucocytes. Evening temperature normal.

26th.—Morning temperature normal. Crescents still present in the blood. No unpigmented parasites. Evening temperature normal.

27th.—Temperature normal. Appearances the same as yesterday.

28th.—Temperature normal. Appearances the same as yesterday.

February 1st.—Crescents still present. Patient discharged to duty.

In this case crescents were present in the blood without fever, and when fever appeared the unpigmented form was evident. While the crescents were present in this man's blood he was quite fit and well. No flagella appeared at any time.

CASE 2.—Private B—, admitted to hospital on the 24th January, 1896. On the 24th his evening temperature was $102\cdot4^{\circ}$ F.; 25th, morning temp. 104° F., evening $100\cdot6^{\circ}$ F.; on morning of 26th normal, evening 104° F.; on morning of 27th $105\cdot8^{\circ}$ F., evening 101° F.; morning of 28th normal. The blood examined on the morning of the 27th contained numerous minute unpigmented parasites, and on the morning of the 28th the same with crescents. Evening temperature of 28th 105° F.

29th.—Morning temp. 101° F. The blood contained unpigmented parasites and crescents.

30th.—Temperature normal. Nothing abnormal noticed except pigmented leucocytes.

31st.—Temperature normal. Pigmented leucocytes.

February 1st.—Temperature and microscopical appearances of the blood normal.

Patient readmitted on the 21st. Temperature at 12 noon 101° F., and at 6 p.m. 101° F.

22nd.—Morning temperature normal. The blood con-

tains numerous unpigmented parasites. No crescents. Some cells contained two parasites. One parasite had a dumb-bell shaped appearance. Evening temperature normal.

23rd.—Temperature at 6 a.m. 99° F., at 9 a.m. 101° F., and at 10.30 a.m. 103·4 F. The blood at 10.30 a.m. contained minute unpigmented parasites. In one cell in which I observed three parasites I saw one of them suddenly resolve itself into two separate bodies. The parasites less numerous to-day than yesterday. This patient refused to take quinine.

CASE 3.—Lance-Corporal C—, has just returned to Sierra Leone from the Ashanti expedition, arriving on March 2nd, 1896. After marching to Mount Auriol, a distance of two miles from the landing stage and about 800 feet above the sea, patient states he had a slight attack of fever the same night, which passed off in a few hours. He came to hospital on March 4th, stating that at 2 a.m. on the 4th he went to the rear, and on returning felt shaky and cold, for about half an hour had headache, afterwards became hot, and then reported himself sick. He says that he never had fever before, not even on the expedition. Morning temperature when admitted to hospital on the 4th, 103·6° F. Patient could retain nothing, and was put in a wet pack for half an hour, after which his temperature fell to 99° F. The blood was examined before wet packing, and was found to contain numerous unpigmented and a few pigmented parasites, mostly oval-shaped, with a ring of hæmoglobin (part of the red cell) surrounding them—the intra-corporal crescent form. The pigment was confined to the centre. Evening temp. 101° F.

March 5th.—Morning temperature normal. Blood contains numerous unpigmented parasites and numerous crescents, some large and others small, with a very little pigment. A few small, rounded, unpigmented bodies were evident in some of the crescents. Pigmented leuco-

cytes. Evening temp. 101.2° F. From this date the temperature remained normal.

8th.—Only crescents.

11th.—Blood examined before patient left hospital. Nothing abnormal, but only a cursory examination could be made.

This case is interesting from the very early appearance of crescents in the blood.

CASE 4.—Corporal T—, after having been in hospital for a week, and apparently sufficiently well to leave, was seized on the night of the 11th March, 1896, with another attack of fever.

March 12th.—Morning temperature 105° F. He was put in a wet pack, and temperature fell to 102° F. The blood contained numerous unpigmented parasites. These parasites were rounded in shape, and their diameter was one sixth of that of a red corpuscle. The colourless protoplasm was peripheral, the transparent centre transmitting the ordinary colour of the red corpuscle, giving the whole parasite a rounded shape. From their periphery small protoplasmic processes were actively projected and retracted; the parasite itself contracted and dilated, during the contraction the size being only about one half that of dilation. One oval-shaped pigmented parasite was observed; a narrow ring of hæmoglobin surrounded it and retained its colour.

Evening temperature 99° F. Blood contains a number of unpigmented parasites. No crescents or other forms were observed.

13th.—Morning temperature normal. The blood contains numerous pigmented and unpigmented parasites; the former contain one or two minute grains of pigment in the periphery, which continued to move for a considerable time. These grains of pigment moved round the periphery, and would suddenly be carried towards the centre of the parasite and back again to the periphery. The parasites were contracting and dilating. The pigmented

forms were about twice the size of the unpigmented. No crescents or oval forms were observed. Pigment-carrying leucocytes were seen.

A specimen was stained by allowing a small quantity of methylene blue to dry on the slide over which the drop of blood was placed. When examined, the central portion of the unpigmented parasites was stained blue, leaving the periphery quite clear. Evening temperature 99.8° F., and going up. The blood contained only a few unpigmented parasites. During the day the patient had taken 40 grains of hydrochlorate of quinine.

14th.—Temperature normal. No parasites visible in the specimens taken.

15th.—Morning temperature normal. Blood contains a few unpigmented parasites. Nothing else abnormal observed. Hydrochlorate of quinine continued in 15-grain doses twice daily.

21st.—Temperature has been normal since the 15th. To-day one parasite only observed, which was slowly throwing out its processes and having two grains of pigment. It was about twice the size of the unpigmented stage.

23rd.—Morning temperature 100° F. The blood contains a few unpigmented forms. Evening temperature 101° F. A few unpigmented forms and crescents.

24th.—Temperature normal. No parasites observed. Up to this time he continued to take 30 grains hydrochlorate of quinine daily. He left hospital on this date.

June 30th.—Returned to hospital with rigors. The blood contained numerous unpigmented forms. This patient had a great many admissions, and had a number of attacks during each admission.

CASE 5.—Private F—, March 16th, 1896. After he had been in hospital for some days it is noted that his temperature on the evening of this day was 102° F. The blood contained pigmented and unpigmented parasites of the size and form described in previous cases. Both

showed the characteristic staining with methylene blue when a small quantity of the stain was added to fresh blood.

17th.—Temperature normal. The blood contains a few unpigmented parasites. A small red cell contained an oval-shaped body with pigment, and between the pigment and the periphery two small spore-like bodies moved about.

18th.—Temperature normal. In a specimen of blood, to which solution of eosin had been added, numerous parasites were observed, and one slightly crescentic body, having a somewhat granular appearance, and tinged with hæmoglobin.

19th.—Temperature normal. The blood contained a few unpigmented parasites, but a prolonged examination was not made. The parasites in this case corresponded in appearance precisely with those observed in the previous cases. The man had the routine treatment with quinine all the time he was in hospital.

CASE 6.—Private E—admitted to hospital on the 27th March, 1896. Evening temp. 103.2° F., going up to 105° F. at 9 p.m. Five grains of calomel and 2 ounces of Mist. Senna Co.

March 28th.—7.30 a.m., temp. 104° F. At 10 a.m. 10 grains of antifebrin and 20 grains of quinine hydrochlorate. 10.30 a.m., examination of the blood. Unpigmented parasites and a spherical body surrounded by the remains of the red cell, with a small mass of pigment in the centre. One specimen was stained by adding methylene blue to the fresh blood, and a parasite observed which almost filled the red cell, and had taken the blue stain. There was a mass of pigment at one part of the periphery. In the unstained specimen, in addition to unpigmented forms there were red blood-cells containing small masses of pigment, the pigment masses consisting of light and dark portions. The clear part of the parasite pushed out pseudopodia-like processes. Whilst one was under observation the red cell gradually disappeared.

29th.—Temperature normal. Many red cells containing parasites spherical in form, and nearly filling the red corpuscle. In some the pigment lay towards the periphery, in others it was central. After being under observation for about an hour a spherical parasite became oval, and the central pigment grouped itself towards the centre, and could be observed moving for several hours. No free crescents were observed.

30th.—Morning temperature normal. One oval pigmented form and three unpigmented forms in one cell. No crescents.

CASE 7.—Private S—admitted to hospital April 1st, 1896, at 5 p.m., comatose. Seen by Surgeon-Captain Hall, who remained with him till 7.30 p.m. I saw him about 8.30 p.m., and found him quite comatose. On touching the conjunctiva there was at first a slight contraction of the orbicularis. Pupils somewhat contracted, no reaction to light; pulse 80, resp. 20. Temperature on admission 103° F., and in the evening 100° F. Breathing not stertorous. Up to 11.30 p.m. he received 60 grains of hydrochlorate of quinine hypodermically, 1 drachm of ether, and two enemata. The patient remained comatose during the night. Morning condition was the same.

April 2nd, morning.—The blood contained numerous pigmented and unpigmented parasites. No crescents visible, but I had not time to examine the peripheral blood very long. At about 1 p.m. there was a sudden quickening of pulse and respiration to 120 and 40. At 4.30 p.m. pulse 170, resp. 60. Still comatose, coma vigil. Temperature rose to 105° F.; condition remained the same till death at 12.40 a.m. on the 3rd.

Post-mortem at 9 a.m. All the organs congested, lymph on surface of brain. Liver weighed 5 pounds. Spleen slightly enlarged, soft and friable, of a dark plum-colour. Brain capillaries contained many pigmented parasites, the pigment being in one small block near the centre of the

parasite; one sporulating form was observed. The affected red cells did not move. Blood from the liver and spleen contained much pigment. One pigmented parasite was observed in the blood from the spleen. I stained a specimen all night in hæmatoxylin, and next morning added a drop of eosin (1 in 1200). Several nuclei of the parasites took on a distinct purple stain, with one or two dark points in the clear part. In the centre of many of the parasites the eosin-stained red cell was visible, and each parasite had a ring of purple round it with one or two dark points.

This patient was in hospital a month before with an ordinary attack of malaria. He returned to duty in a few days. For two days previous to his last admission to hospital he had a slight attack of fever, but wishing to finish shooting in a rifle competition, he remained for about nine hours on each of these days at the rifle range, and very probably this prolonged exposure led to a fatal result. This case shows the necessity of absolute rest, however trivial the attack of fever may appear.

CASE 8.—Corporal K— admitted to hospital on May 19th, 1896; temp. 101° F. Patient states that fever began at 4 a.m. He has had many previous attacks of fever. The blood contained a number of unpigmented parasites, and crescents with the usual appearances, pigment being always in the centre except in one in which it was scattered. In some there was only a very small quantity of dark pigment without the appearance of the central body in which I usually observed the pigment grouped. Evening temperature normal.

May 20th.—Morning temperature normal. The blood contained several small round pigmented forms, in the centre of which the red cell was visible, and they every now and then contracted and expanded. The pigment was in active movement. Numerous crescents.

21st.—Temperature normal since first day. Crescents less numerous. No other forms.

25th.—Temperature normal. Patient discharged. Crescents still present. No other forms.

CASE 9.—Private F—, admitted on the evening of June 26th, 1896. Temp. 104·6° F.

June 27th.—Morning temp. 99·2° F. Evening temperature normal.

28th.—Morning temperature normal. Evening 101° F.

29th.—Temperature normal. He had twenty grains of quinine hydrochlorate daily during this time.

Microscopical examination of the blood.—Very many red cells much enlarged, paler than normal, and containing minute numerous pigment granules. A few contained what looked like definite amoeboid bodies. There was distinct movement of the pigment granules. In the unstained specimens I did not observe any of the forms seen in the cases of pernicious malaria which I have described, but in the stained specimens I saw a small parasite in the red corpuscle with one very small pigment granule. This body resembled a form described in the pernicious cases.

This is the first case of tertian fever I have seen at Sierra Leone. Patient states he had several attacks of malaria in Jamaica. He came to the coast in the beginning of 1894, and it is legitimate to infer that the attack may have been a recurrence of the malaria acquired in Jamaica. (I make this remark with all deference.)

This man's case differed from the others in the accented rigors which ushered in the attack. It will be noted that while he had fever on the 26th and 28th, on the 27th he was free from fever, and the fever yielded rapidly to 20-grain doses of quinine daily. He was kept under observation for some time, and there was no relapse. These features agree with those which are characteristic of tertian ague, and the microscopical examination of the blood showed that the case was a true tertian. The comparatively large size of the parasite, still more the very much enlarged red cells, which to a great extent had lost their colour, sharply differentiated the microscopical

appearances from those which I have described in the pernicious cases, and harmonised completely with the description of the parasite of tertian fever, as described by Golgi and others.

CASE 10.—Private H— has been in hospital several months, suffering from heart disease, and has had several attacks of fever during that time. He states that the attack usually begins with shivering, but the attack this morning commenced without rigors. Temp. $99\cdot4^{\circ}$ F. Vomited once.

Examination of the blood shows many unpigmented forms and many crescents. An appearance which I observed in this case, and which I had never before observed, deserves special mention. A red cell was nearly filled with a parasite, the parasite consisting of clear protoplasm on the periphery, and in the interior a number of small irregular-shaped bodies in close contact, presenting a somewhat dendriform arrangement. While under observation this body, which was crescent-shaped, first enlarged, and then the small bodies which I have described were observed to oscillate and arrange themselves in a ring form, after which a bulging took place in the side of the crescent, and these bodies appeared to endeavour to become free, but apparently the wall of the crescent would not yield. The crescent swelled to about twice its original size, and the small bodies which it contained scattered themselves irregularly in its interior, and then ceased to oscillate. I do not wish to associate this solitary observation with any general law, but in connection with Grassi and Feletti's theory of the sporulation of the crescent body I have thought it well to place it on record. It was a solitary example seen during a series of observations of the crescent form, of which I must have watched some hundreds closely. At 2 p.m. temp. $102\cdot8^{\circ}$ F.; 6 p.m., temp. $99\cdot4^{\circ}$ F.; 9 p.m., normal. A number of crescents with a nipple-shaped projection were observed in the blood.

July 9th.—Fever began shortly after 10 a.m. Tempera-

ture at 4.30 p.m. 103.6° F. I observed an oval-shaped parasite with a pigment mass similar to that seen in crescents. It filled the red cell, from which the colour had entirely disappeared. It contained three distinct spores with a distinct nucleolus in each spore. The pigment was in clumps. Close to it, but not touching this body, was a patch of pigment to which a spore was adherent. This appearance is the nearest approach to what might be a crescent in sporulation which I observed. Several pigment-containing leucocytes were observed, the pigment granules being in motion.

11th.—2 p.m., temp. 102.2° F. ; 6 p.m., temp. 101.4° F. ; 9 p.m., temp. 101.4° F.

12th.—Temperature normal. Patient then passed out of my observation. During the attack quinine was given in the usual doses.

CASE 11.—Private M—, admitted to hospital July 10th, 1896, suffering from fever.

The blood contains pigmented and unpigmented parasites. The pigment is present in the form of one or two grains situated towards the periphery. The portion of the red cell present in the centre of the parasite sometimes consists of two parts. At 2 p.m. temperature 100.8° F. ; at 6 p.m., 101.8° F. ; at 9 p.m., 104° F.

11th.—Temperature at 9 a.m. 103° F. I stained a fresh specimen with Plehn's fluid, which caused the periphery of most of the parasites to become motionless, while the central mass appeared as a dark spot. The nucleolus became evident on staining some of them. In one unpigmented parasite there were two nucleoli deeply stained and moving most actively. The periphery of this parasite threw out processes, and it moved about the red cell, an appearance which I had not before observed. In the centre of many of the parasites three or four minute dark bodies moved rapidly. They were probably minute pigment granules. No crescents evident. A special appearance which was observed during this examination

should be noted. In a parasite which filled one half of the red cell, and which was oval and distinctly contoured; a block of pigment was situated at one end of the parasite, whilst a single solitary spore was observed at the other. The granules of pigment, although massed together, were in active movement. The appearance of the pigment and the shape of the parasite are very suggestive of a crescent formation (the *Laverania* of Grassi and Feletti). If this were the case, it would afford additional proof that the crescent form can produce a spore, but I do not insist on this interpretation. The appearance is not entirely inconsistent with the interpretation of a sporulating form of the pernicious parasite. Without insisting on any special interpretation, I simply put the fact on record. At 2 p.m. temperature 101.8° F.; at 6 p.m., 100.4° F.; at 9 p.m., 99.8° F.

12th.—Temperature at 7.45 a.m. 99.8° F.; at 10 a.m., 101.4° F.

13th.—Minute dark points no longer visible in the parasite. Few parasites visible. A parasite was seen throwing out processes on the blood-cell.

Of these 11 cases all were negroes, with the exception of Case 4, a white man.

If we exclude a case of tertian fever (the only one observed amongst about 400 cases), it will be seen that in the malarial fevers of Sierra Leone the only parasitic forms observed in the peripheral blood are a very small amoeboid organism, usually ring-shaped, a slightly further advanced stage in which a little very fine pigment can be detected on the periphery, a further stage in which the pigment is massed towards the centre of the parasite, which has enlarged to about twice the original size (only observed a few times), and the crescent forms fully developed, and in the partially developed intra-corpuscular forms. In the amoeboid and pigmented forms nuclear elements can be observed. Nuclear elements were not seen in the crescents, but a differentiated central portion in which the pigment

accumulates could be made out. In one case appearances were observed which could be explained by assuming the accuracy of the theory of Grassi and Feletti that the crescent body may form spores, but I do not desire to lay much weight on a solitary observation. In blood taken from a vein in the brain of a fatal case I observed a sporulating form in which both the central pigment and the spores were distinct. I never saw the so-called rosette form in the peripheral blood. I never observed the quartan forms described by Golgi, and only once the characteristic appearances of the tertian parasite of that observer.

In order to compare the parasites which I saw with the quartan and tertian forms of Golgi, I was obliged to get specimens containing these forms from Europe. Dr. Thin was kind enough, at my request, to send me preparations containing the tertian and quartan parasites. These were very distinct, and I was able to satisfy myself that no such appearances (always excepting the solitary case of tertian) were present in the cases which I examined. Is there anything in my description of this parasite which can be considered evidence that the parasite of malaria in Sierra Leone is a distinct species? I find that there is not. If my description and the drawings which accompany this paper are compared with the description and the drawings which illustrate the work by Marchiafava and Bignami, it will be seen that the Sierra Leone forms correspond in every essential particular with the parasites which those authors describe as being found in the so-called summer-autumn fevers of Southern Italy. The small amoeboid ring-shaped forms and the small forms with very fine pigment find their exact countertypes in the descriptions of the Italian authors. The "ottonati" or brassy forms described by them I did not observe. This is the only particular in which my description appears to differ from theirs, and in this connection it may be noted that Thayer and Hewetson in describing the summer-autumn fevers at Baltimore also find many of the so-called brassy-red corpuscles.

As regards the ætiology I can add nothing to what has already been written on this subject.

Expeditions into the interior of Sierra Leone, which are generally limited to two months on account of the difficulties of transport, are followed by a large increase in the cases of malaria. After the regiment has returned to Sierra Leone, fever very soon attacks officers and men, more especially the former, and a fatal result is by no means rare.

Among the officers of the Sierra Leone Frontier Force, who have to spend most of their fifteen months' tour in the interior, the mortality is higher than is the case with the officers of the West India Regiment who serve for a year on the coast.

Mosquitoes are present before and during the rainy season. There are so few that mosquito nets are hardly necessary. Many of the officers are bitten by these mosquitoes, some rather severely, but I do not know of a single case having been followed by fever.

It may be interesting to state that I have found examination of the blood of value diagnostically in cases brought into hospital comatose. There are cases in which it is important to eliminate other causes of coma, such as apoplexy and alcoholic poisoning. Persistent headache and cases of subacute rheumatism have been generally returned as malarial, but I have found by microscopic examination of the blood that many of these cases, particularly the latter, are frequently not caused by malaria. In all cases of malaria I found the parasite, and therefore its absence may be held as conclusive of the case being non-malarial.

For treatment I shall simply refer to the routine administration by most medical officers on the coast, of calomel, antipyretics, and quinine. Wet packing is extremely useful in cases of persistent vomiting. I obtained no benefit from boric acid in 30 grains thrice daily, or Liquor Hydrarg. Perchlor. in drachm doses thrice daily. Arsenic appeared to be of service in one case of malarial fever which lasted

for several weeks. I did not employ any of these three drugs for a lengthened period.

Quinine should be given early and in large doses (15—20 grains), repeated every two to three hours until the temperature is normal.

Absolute rest is necessary in every case, however trivial the symptoms may appear.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 99.)

DESCRIPTION OF PLATE XXIII.

Figs. 1—22 represent the appearances observed in the fresh blood.

Fig. 1.—Small ring-shaped parasite. Fig. 2.—Four such parasites attacking one red corpuscle. Figs. 3, 4, and 5 show the parasite in the active motile stage. Figs. 6, 7, and 8 show the parasite in the stage in which pigment formation begins. Figs. 1—8 are blood taken from the finger.

Figs. 9—12 represent red blood-corpuscles from a drop of blood taken from the surface of the brain in a fatal case, about eight hours after death. Central pigment can be seen in all four. In Fig. 10 it appears divided into two portions. In Fig. 12 five separate spores can be counted.

Figs. 13—22 represent various appearances shown by the crescent form. In Fig. 13 the crescent nearly fills the red corpuscle. Fig. 14 shows a round crescent nearly filled with pigment, the collection of pigment being unusually large. Fig. 14*a* represents the parasite shown in 14, after it had undergone change, the pigment collecting in the centre. This change occurred whilst the parasite was under observation. Fig. 15.—A crescent with central pigment, filling about half the red corpuscle. Fig. 16.—Larger crescent than 15, almost filling the red corpuscle. Figs. 17, 18, and 19 show various types of the crescent parasite.

Figs. 20, 20*a*, 20*b*, 20*c*.—Transformations of the parasite observed under the microscope, which is described at p. 231. The first form of the parasite observed is shown in Fig. 20; the other three figures show the gradual transformation which took place. Fig. 21 represents what the author considers to be a form suggestive of spore formation in a crescent (see p. 232). There was no doubt as to the crescent nature of this body, and the pigment blocks and spore-like forms were separate and distinct in its interior. Close to the crescent, adhering to some pigment, was a free spore.

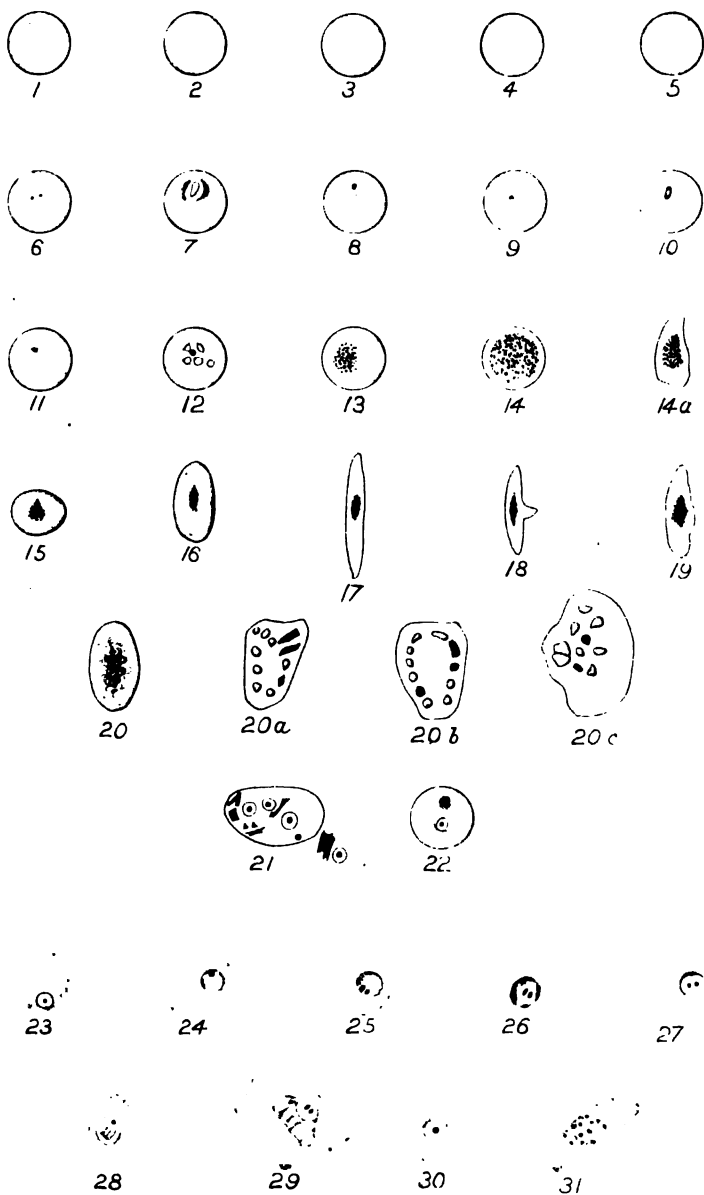




Fig. 22 shows a small crescent body inside a red corpuscle, with a pigment block at one end, and a small circular body with a central point at the other end—suggestive of a spore. This spore-like body was identical in appearance with the three spore-like bodies shown in Fig. 21.

Figs. 23—31 represent appearances in stained preparations.

These preparations were stained at Sierra Leone, and were drawn by Mrs. Danielsson.

Fig. 23 shows the minute nucleolar body. Fig. 24 shows a similar nucleolar body on the periphery. Fig. 25.—No separate nucleolar body was observed. Fig. 26.—The deeply stained nucleolar body is surrounded by an uncoloured portion, whilst the periphery is stained blue. Figs. 27, 28, and 29 show similar phases. They were observed in one preparation in the same field of the microscope adjoining each other. In Figs. 26, 27, and 29 there were two nucleoli in each parasite. In Figs. 26, 27, and 29 the colourless space surrounding the nucleolus, which has been described by Mannaberg as the nucleus, is very distinctly observed. Fig. 30 shows the young form of the parasite in a case in which there were many crescents in the blood. Fig. 31.—Fully developed crescent form from same preparation as Fig. 30.

In none of the parasites represented from 23 to 30 had the development reached the stage of pigment formation.

Figs. 1—22 represent as near as possible the size of the elements drawn as seen by $\frac{1}{2}$ oil immersion lens and Zeiss No. 8 eye-piece. Figs. 23—29 were purposely drawn to a similar scale.



PULSATING EMPYEMA

(EMPYEMA PULSANS)

BY

SAMUEL WEST, M.D.

Received November 2nd, 1896—Read March 23rd, 1897

EMPYEMATA may pulsate, but distinct pulsation is certainly a very rare phenomenon. When Comby¹ wrote his paper in 1882 he did not succeed in finding more than about thirty cases recorded.

Wilson² in 1893 brings up the number of published cases to sixty-eight, but the increase in numbers of recent years is chiefly due to the larger number of cases recorded in which there has been a general shock, more or less distinct, felt over a considerable area of the side.

The fact remains that distinct pulsation is very rare, but the condition is one which always attracts attention, on account of the difficulties of diagnosis to which it may give rise.

The pulsation is of two kinds :—(1) A general shock or impulse communicated to a considerable area of the chest walls. This has been called intra-pleural pulsation. (2) Expansile pulsation localised in an external tumour. This has been called extra-pleural pulsation.

¹ 'Arch. gén. de Méd.,' Nov., 1883, "Thésis d'empyème pulsatile."

² 'Trans. Assoc. Amer. Phys. Philad.,' 1893, viii, 195.

The pulsation is sometimes visible, but often only to be felt by the hand. It occurs, with but few exceptions, on the left side ; thus of sixty-six cases it was on the right side in five instances only.

The pulsation is synchronous with the heart, and almost invariably systolic in time, though there are three cases in which it has been stated to be diastolic ; two recorded by Macdonald¹ and one by Stokes.²

The condition may be met with in either sex, and at any age. It is more common in males, in the proportion, it appears, of four to one, and in adult life between the ages of twenty and thirty-five. The youngest recorded case was two years, and the oldest fifty-seven. It is rare except with empyemata, and then only with those of long duration ; but it has been described also with serous effusion and with pyo-pneumothorax.

Intra-pleural pulsation.—When the pulsation is intra-pleural it is occasionally visible, but usually only to be felt by the hand. Usually it is widespread and felt over the lower part of the chest, especially in the axilla, as the patient is lying in bed. Occasionally it is felt higher up and more in front. It may be limited to one or two intercostal spaces in the supra-mammary region, or be more diffuse and extend outwards from the sternum to the middle, or posterior, axillary line, and downwards from the third rib to the lower part of the axilla. In these cases, especially where the pulsation is felt high up, it is probably analogous to that which is felt in the abdomen in ascites, and partakes of the character of a fluid wave.

Intra-pleural pulsation may occur with any kind of effusion, chiefly, it is true, with empyema, but also with pyo-pneumothorax, and sometimes even with serous effusion. Of this latter Traube³ recorded the first case, and several others have been described in recent years.⁴

¹ 'Dubl. Journ.,' 1854, xxv.

² 'Dis. of Heart and Aorta.'

³ 'Ges. Beiträge,' 1871.

⁴ Fraentzel, Ziemssen, iv, 2nd, p. 355.

Extra-pleural pulsation.—Extra-pleural pulsation, *i. e.* pulsation limited to an external tumour, is met with in empyema only. It occurs when the empyema is pointing, and if the empyema is pointing in more than one place, may occur in two or three of the tumours. Thus, of the cases recorded, in three instances there were two, and in two instances three, external tumours, all of which pulsated.¹

In many of these cases the tumour and the pulsation in it may vary with respiration, the tumour becoming larger and the pulsation more distinct with expiration, and less so with inspiration. Sometimes both the tumour and the pulsation in it may be made to disappear by pressure, that is to say, the pus may be returned within the thorax.

These pulsating tumours may be met with in any part of the chest where an empyema points, but most of them somewhere near the heart, and of course this will be their most likely position if the empyema is localised. Thus they may occur above the præcordial region, but are found more commonly in the mammary region or near the apex, and occasionally even immediately over the præcordium in front.

Where there is more than one tumour they are usually close together, but they may be widely separated, *e. g.* one tumour may be near the heart and the other close to the spine (Macdonell, Wilson Fox).

Occasionally, if the empyema has tracked, a pulsating tumour may be found in a very unusual place, *e. g.* in the loin. Thus Owen Rees² records a case in which the empyema tracked to this spot, and the post-mortem showed that it was due to an empyema; the pulsation in this case being transmitted, not from the heart, but from the abdominal aorta, with which the abscess was in close relation. A similar case is recorded by Müller.³

Diagnosis.—The diagnosis may be very difficult, but the difficulties are not quite the same in intra- and extra-pleural pulsation.

¹ Wilson, *loc. cit.*

² 'Brit. Med. Journ.,' Aug. 21st, 1858.

³ 'Berl. klin. Wochenschr.,' Jan. 22nd, 1872.

Intra-pleural pulsation does not, as a rule, present any great difficulties, for it is rarely met with except with large effusions, and then the nature of the case is evident. If it should be limited, however, to the anterior part of the chest or to the region of the heart, it might need to be diagnosed from a dilated or hypertrophied heart on the one hand, or from an aneurism of the heart or of the aorta on the other. It is not, however, in these cases that the difficulties occur, or if they do that they are of any great practical importance.

The association of the two conditions, viz. of a pulsating pleural effusion with an aneurism of the aorta, is very rare. I only know of one instance, viz. that recorded by Stokes,¹ and in that case the aneurism was very small.

The diagnosis from pulsating pneumonia and a pulsating intra-thoracic new growth, as suggested by Graves, is very unlikely to make difficulty, for these cases are, to say the least, extremely rare. I have never seen a marked instance of either. As to the existence even of pneumonia pulsans I am very sceptical.

With *extra-pleural pulsation*, i. e. pulsation in an external tumour, the difficulties are also not, as a rule, great, for the physical signs indicate the existence of a large empyema which is pointing.

If the empyema be localised, especially in the neighbourhood of the heart, the difficulties of diagnosis may be greater. If the tumour is near the apex of the heart or over the lower part of the præcordial region, the question of cardiac aneurism might arise; yet aneurisms of the heart hardly ever cause an external tumour in this position. They develop rather in other directions within the thorax. I have, it is true, seen one case of cardiac aneurism causing an external swelling. This occurred on the right side of the chest, between the nipple line and the lower part of the sternum, but there was no doubt about the diagnosis.

Where the swelling is higher up, and especially if it be in the first and second intercostal spaces, the chances of

¹ Loc. cit.

aneurism would be greater. Thus, in Topham's¹ case, in which there was a pulsating swelling in the third intercostal space close to the sternum, an aneurism was diagnosed, and the patient was kept in bed for about three years, when a discharge of pus took place, the tumour disappeared, and the patient got well.

In Aran's² case an abscess in the upper part of the mediastinum caused an external swelling. This was not connected with the pleura. It discharged, and the patient recovered. And in Vidal's³ case a similar pulsating swelling over the lower part of the sternum proved to be also due to a small abscess in this position; but this patient was suffering from double empyema, though no direct communication could be traced between the mediastinal abscess and the pleura.

Localised empyemata in the neighbourhood of the heart are very rare, and even when they occur do not often cause external pulsation. In a remarkable case in which the empyema was on the right side of the chest between the sternum and nipple, so that the dulness was continuous with that of the heart, though the empyema was in close apposition with the right side of the heart there was no trace of pulsation communicated externally. On the other hand, even when the tumour is due to an aneurism, it need not necessarily pulsate, for the interior may be filled with clot.

The difficulties of diagnosis are greater between a local abscess of the chest wall which pulsates, and an aneurism, than between an aneurism and an empyema, because, in the majority of cases, the signs of empyema are so characteristic.

Superficial abscesses are most likely to give difficulty when they are seated somewhere near the apex of the heart, where the pulsation is forcible and the intercostal spaces fairly large, and they are still more likely to pulsate if there be pus beneath the ribs as well as outside

¹ 'Lancet,' 1878, i, p. 756.

² 'Bull. de Soc. Méd. des Hôpitaux,' 1858, xv, 90.

³ 'Bull. de la Soc. Anatom.,' 1854, p. 243.

them, as, for instance, with a periosteal abscess due to caries of a portion of the rib in this position. I have seen one instance of this kind in which the pulsation was so marked that no one dared to interfere with the tumour. Ultimately the skin burst, pus was spontaneously discharged, and the tumour entirely disappeared.

I think, in cases of this kind, a small puncture with a very fine needle, for the purposes of exploration, would do no harm whatever, even if the tumour proved to be an aneurism, and of course it would at once clear up the diagnosis if pus was obtained. Over such a tumour a murmur would not, as a rule, be present, as it might be in the case of an aneurism; but then a murmur is not necessarily present even if the tumour is aneurismal, and, as stated, some of these external aneurisms do not even pulsate.

Explanation.—Pulsation being, as a rule, absent in effusions of all kinds, of whatever size they may be, it is evident that its occurrence requires peculiar conditions. There appear to be three different kinds of pulsation, and the same explanation will not fit them all.

1. There is, first of all, the general shock felt over the lower part of the chest, usually in the posterior axilla and behind. This is found only with very large effusions, and necessitates considerable intra-pleural pressure. It is the same thing as the general shock which is transmitted sometimes to the chest walls, from the heart through a solid mediastinal or pulmonary tumour, or through massive pneumonia.

2. There is next that form of pulsation which is met with chiefly in front, and in the upper part or mammary region of the chest, and which does not, as a rule, extend beyond the anterior or mid-axillary line. It is to this group that most of the recently recorded cases appear to belong. This kind of pulsation is, I think, strictly analogous to that which we meet with in the abdomen in ascites, and is due to the propagation of a fluid wave from the heart to the surface of the fluid. It is felt where the surface of the fluid would be as the patient is lying in bed

upon the back, and would, I think, very likely alter its position if the patient sat up, though I do not know that this has been actually described. As in the abdomen it is to be connected with a certain laxity of the chest walls, for fluctuation is not nearly as distinctly felt in the abdomen when the walls are tense as when they are relaxed. For this reason this form of pulsation occurs usually with empyemata, and with empyemata of some duration, though it might possibly be met with in other forms of effusion, and also with pneumothorax.

3. In the third form of pulsation, viz. that in which the pulsation has more of an expansile character, whether it be felt over a large portion of the chest or limited to an external tumour, the explanation, I think, is different.

This last kind of pulsation is more like that which we get in a large abscess, and which we speak of as "surgical fluctuation;" and in order that this should be obtained the fluid must be contained within some more or less resistant walls, *i. e.* must be under some pressure.

Wherever there is a large collection of fluid in the pleura, on the left side especially, it is obvious that the pulsations of the heart must affect it; but in most cases the pulsations spend themselves upon the least resistant part of the abscess walls, viz. upon the incompletely collapsed portion of the lung or upon the mediastinum, or if pneumothorax be present they exhaust themselves in compressing the air in the pleura.

In order that the pulsation should be transmitted distinctly to the chest walls or to an external tumour, it is necessary that the other parts of the walls of the pleura should be rigid and resistant, so as to enable the pulsations to be transmitted to the thoracic walls, *i. e.* to what are usually the more resistant parts. In the case of a general empyema, *i. e.* of an empyema occupying the whole pleural cavity, this kind of pulsation presupposes the rigid fixation of the internal walls, viz. of the heart and mediastinum, and of the lung also. This is exactly the condition which has been on many occasions described.

Of this Comby's case is a good instance. On post-mortem examination the heart was found to be rigidly fixed to the right side of the chest, and the lung to be adherent all along the mediastinum from the sternum to the spine, as well as at the apex, and to be in a condition of extreme fibroid induration. The empyema in this case was a large one and the pulsation distinct; 80 oz. of pus were removed, and the pulsation disappeared, until the fluid re-accumulated, when it returned.

For this kind of pulsation, therefore, we require that the internal walls of the pleura, the mediastinum, and lung should offer almost as much resistance to the pulsation of the heart as the chest walls, and such conditions are not likely to be common.

Where the empyema is pointing, that is to say, where the pus has made its way through the chest walls, and there is a fairly free communication with the pleura, the resistance is very much less, and we should expect, *a priori*, that pulsation would occur in these conditions much more frequently. This we know to be the case, for this kind of expansile pulsation is met with most frequently with external tumours, that is to say, where an empyema is pointing.

In the same way, where the empyema is localised pulsation of this kind necessitates considerable induration of the walls surrounding the abscess, and probably a considerable amount of intra-pleural pressure.

These cases are interesting chiefly on account of the difficulties of diagnosis, but the diagnosis once made the treatment is that of empyema, *i. e.* they may be tapped or opened as may seem desirable, and with success.

It has been stated that pulsating empyemata are, almost without exception, fatal. This is an over-statement of fact, and is far from true. Thus, in Wilson's cases, out of forty-six in which the result is stated, twenty-four recovered, *i. e.* about 52 per cent.

The cases which are generally fatal belong to the last group which has just been described, viz. that in which

there is expansible pulsation of a very marked kind. These cases are fatal, not because the empyema pulsates, but because of the peculiar conditions, viz. the fixation of the heart and the changes in the mediastinum and lung, which prevent cure. Of the other cases the great majority recover.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 104.)

COMPOUND DEPRESSED FRACTURE OF THE SKULL

CEREBRAL ABSCESS; HERNIA CEREBRI;
RECOVERY

WITH A CONSIDERATION OF THE SUBJECT OF HERNIA
CEREBRI BASED UPON ONE HUNDRED AND
NINE COLLECTED CASES

BY

R. LAWFORD KNAGGS

ERRATA.

(Mr. Knaggs' Paper.)

Page 272, Case 14, 1st line, should read "From 'Medical Commentaries,' " &c.

Page 273, Case 20, 2nd line, should read "Man aged 24," &c.

Page 282, Case 49, 1st line, should read "London Medical Repository," &c.

Page 283, Case 50, 4th, 5th, and 6th lines, should read "Symptoms occurred in four days. As granulations advanced and loose bones came away hernia cerebri protruded until it overlapped the entire wound."

Page 291, Case 78, 15th and 16th lines, February 12th, &c., "2½ inches" should read "2¼ inches."

Case 79, 1st line, "p. 336" should read "p. 337."

Page 292, foot-note, 1st line, "facts" should read "cases."

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(COMMUNICATED BY MR. BRYANT)

Received November 2nd, 1896—Read April 13th, 1897

JOSEPH G—, aged 31, fell from a coal stage upon his head on September 11th, 1894, in the morning, and was admitted soon after into the Leeds Infirmary. In Mr. Mayo Robson's absence he came under my care. He was suffering from concussion and a compound depressed fracture of the left parietal bone, with a lacerated skin wound, and there was, besides, right facial paresis. This was permanent, and it remained doubtful if it had not existed before the accident. Consciousness was completely

recovered soon after admission. Under ether the wound was enlarged, and all the depressed bone and loose fragments removed, an aperture about three inches by two being left. There was a slit in the dura mater about one third of an inch long, and on holding its edges aside a clean, small, incised wound was seen on the surface of the brain. Cerebro-spinal fluid escaped freely from this opening, but after closure by a catgut suture the flow almost stopped. The wound was then closed, every care being taken to render it aseptic. The temperature was 100° on the 11th and 12th, and then fell to normal, and, with very slight variations, maintained a normal course during his stay in hospital. The wound suppurated, and a slough at the bottom of it took some time to get rid of, but by November 11th it had healed. In other respects the patient's progress had been satisfactory till October 3rd, when he did not feel well; on the 4th he had two, and on the 5th one convulsive seizure affecting all his limbs, but more particularly those on the right side. He was ordered half-drachm doses of bromide of potash, and no further recurrence took place. On October 10th the right arm was markedly weaker than the left, and his articulation was affected. On November 17th his defect is described: "He knows what he wants to say but his difficulty is to say it."

By the end of November, when he was discharged, the paresis of the arm had largely recovered and his speech was better, but a blurring of three quarters of the outline of the right optic disc had persisted unchanged for a month.

On December 25th he had three fits, and on January 10th three more. His speech was distinctly worse on January 16th, and his pulse 60 (Mist. Pot. Bromid.). On January 21st the soft parts over the gap in the skull were pushed out about a quarter of an inch, and he complained of pains at the back of the neck. He improved for a time, but on February 4th again complained of pains over the whole head and the back of the neck, and on the

18th he was readmitted. The protrusion over the opening in the left parietal bone was tense, semi-fluctuating, and pulsated. It measured $3\frac{1}{2}$ inches by $2\frac{1}{2}$, had an elevation of three quarters of an inch, and corresponded with portion of the motor area. The pain in the head and neck varied in severity. There was double optic neuritis, more advanced in the left eye. Pulse 60, temp. 97.5° . Urine contained a trace of albumen. Dynamometer : right, 60 lbs. ; left, 56 lbs.

On the 23rd he had a bad night, the pain necessitating several doses of chloral. The following morning he became semi-comatose, and his arms, especially the left, were rigid. Soon after he was found by the nurse at the point of death, and our able resident surgical officer, Mr. Walter Thompson, entering the ward at that moment, explored the swelling at once and drew off some pus. Then with his pocket-knife he made an incision into the tumour, and a quantity of matter, estimated at three quarters of an ounce, spurted out to a considerable distance. Artificial respiration was at once commenced, and in a few minutes the patient came round.

Shortly after, I found the healthy surface of the brain protruding through a widely gaping incision in the dura. There was a small laceration or cut on the exposed convolution. In the afternoon the patient had recovered some consciousness, but his breathing was slightly stertorous and deep. The protruding brain being more prominent and very fluctuant, I explored it to a depth of $1\frac{1}{2}$ inches. McEwen's trocar seemed to enter a cavity and some pus came away in the groove at the end, and a bead of pus escaped on its withdrawal ; but no collection being reached, nothing further was attempted. The head had been shaved and a gauze dressing applied, after careful efforts to render the parts aseptic. At 10.30 p.m. intelligence had returned, and there was marked paresis of both right arm and leg.

He continued to mend, power slowly returning to the right limbs. On February 27th the hernia cerebri, which

was situated on the centre of a general protrusion of soft parts corresponding to the aperture in the bone, measured 2 inches by 1 inch, and had a height of 1 inch. It had been gradually enlarging. On March 10th it overlapped the scalp at the lower part, and any attempt to diminish it by pressure caused it to flatten out. The patient's mental condition, which before and since the operation had been very dull, was now much improved. He read his paper and could appreciate a joke.

After this the right grasp became very feeble (Dyn.: 6 lbs., 11 lbs., 9 lbs., 14 lbs.). The weakness seemed to be chiefly in the forearm muscles, as the finger movements were feeble and uncertain, but he could exert a considerable pulling or pushing force.

The hernia became covered with granulations, but did not increase. On April 5th it first showed signs of beginning to shrink. On the 13th the patient had a fit when his friends were visiting him. It began by the right arm being thrown out. On the 29th, a sore throat and a rise of temperature accounted for the hernia, which had been slowly diminishing, puffing up to its original size, but it subsided in a day or two with the disappearance of the throat condition. The shrinkage continued, and by May 18th skin was beginning to spread up over the sides. There is little more to add. The tumour completely disappeared, and on July 7th the wound was healed and a depression instead of a fulness existed over the bony aperture. When he went home on August 3rd, his general condition was satisfactory in every respect; the optic neuritis was clearing up, his speech, though better, was still slow and hesitating, and for some time he had been making himself useful in the ward (Dyn.: right, 50; left, 60).¹

When last seen, in July, 1896, he was in good health,

¹ I should like to express my obligation to Mr. Walter Thompson, Mr. A. S. Robinson, and Mr. H. Clough for the care and attention they gave to the case and for many of the notes. Mr. Thompson's brilliant promptitude deserves my grateful recognition.

DESCRIPTION OF PLATES XXIV AND XXV.

**Compound Depressed Fracture of the Skull; Cerebral Abscess;
Hernia Cerebri (R. LAWFORD KNAGGS).**

PLATE XXIV

Fig. 1.—March 16th.

Fig. 2.—About March 16th.

PLATE XXV

Fig. 3.—May 21st.

Fig. 4.—After leaving hospital.



Fig. 2.



Fig. 1.

Allard, imp.



Fig. 4.



Fig. 3.

Adlard, imp.



except that he had a fit every eight weeks. He was following some labouring employment, but his general condition was not materially altered.

The uncertainty that seemed to exist with regard to the most suitable treatment of hernia cerebri, an uncertainty that is well illustrated by the various methods that have been employed, induced the writer to investigate the subject.

The 109 cases upon which the following paper is based are in no way selected, except that those from German sources have necessarily been excluded from a want of knowledge of the language.

Conditions antecedent to Hernia Cerebri.

1. Compound fracture of the skull, frequently comminuted and usually depressed. (The membranes were probably injured in all, and in most cases the brain also) . . . 80 cases.
2. Compound depressed fracture, where the membranes were intact, and subsequently gave way (Cases 11, 12, 22, 32 ?, 72, 96, 99) . . . 7 cases.
3. Simple fracture. The dura mater and brain were injured by a fragment (Case 41) . . . 1 case.
4. Fracture of inner table (Case 47) . . . 1 case.
5. Scalp wound (Cases 17, 84) . . . 2 cases.
6. Necrosis—

(a) Syphilitic (Cases 90, 102)	. . .	}	5 cases.
(b) Traumatic (Case 78)	. . .		
(c) "Disease of Bone" (Cases 55, 89)	. . .		
7. Injury to brain, unprotected by a bony covering (Case 25) . . . 1 case.

In the remainder of the cases, information as to the exciting cause is absent.

The Intra-cranial Conditions predisposing to Hernia Cerebri.

1. *Increased tension.*—The existence of this is sufficiently obvious, but it would probably not lead to hernia cerebri if it were not associated with the following.¹

2. *A softened or diffuent state of a portion of the cerebral substance.*—This morbid condition would appear to be essential. It may be local, involving the protrusion and the adjacent portion of the brain, or it may be diffuse, involving large portions or the whole of a hemisphere. An area below the surface may be affected, and as it is moulded by the increased pressure, a crust of normal brain tissue may be pushed before it; thus the exterior of a hernia cerebri may be firmer than its centre (Cases 21, 90).

The softening, too, may be so extreme that the cerebro-spinal fluid can open a track through it from the lateral ventricle to the surface (Cases 24, 96, 109).

In twenty-seven of the *abstracted* cases, direct evidence as to this softened state is forthcoming, and with a single exception this is post-mortem observation. Of the other fatal cases no post-mortem was made in twelve, and no notice is taken of the consistency of the brain substance in eleven. There remain three more. Two give us no help. They were recorded by Abernethy, who was responsible for certain views as to the causation of hernia cerebri. In the third (Case 62, recorded in 1830) it is stated that there was no alteration in the consistency of the brain; still, in that case an abscess containing three ounces of pus had been evacuated before death, so the accuracy of the observation is perhaps open to doubt.

3. *Inflammation of the cerebral substance.*—The softening of the brain tissue is a consequence of its inflammation. The increase in bulk, without which the hernia would not

¹ In connection with this point, attention may be drawn to those cases where the skull-cap is opened for brain tumour. Here the bulging of the brain into the aperture is hardly to be described as hernia cerebri, by which term we understand rather a protrusion.

occur, is due to inflammatory exudation, which leads to a general softening of its tissues, and their replacement by granulation tissue, if the process is sufficiently prolonged. Where the brain has been extensively implicated, the hernia has usually been large, and, when removed, rapidly reproduced (Cases 98 to 101). This might be expected. More than one case is to be found in the appended list, in which after removal of the hernia or sloughing of its surface, a serous fluid has poured away from the exposed portion for a considerable time, and sometimes with relief to the pressure symptoms (Cases 17, 61, 64). In these cases the discharge was almost certainly the inflammatory exudation, and not cerebro-spinal fluid.

The *causes* of the inflammation are—

- (1) Direct injury to the brain itself ;
- (2) Traumatism, with sepsis added, and
- (3) Sepsis alone ;

and as a *consequence* of it, the functions of the area involved are affected, being weakened, or in complete abeyance, and in the worst cases even destroyed more or less completely.

When the motor area is affected, the extension of the disease may be followed as fresh symptoms gradually supervene, and its recession may be traced as symptoms in order decline and disappear.

An extension of the inflammatory cedema to the neighbourhood of the iter may lead to the occlusion of a passage through which the inflammatory serum discharged into the ventricles is usually carried away, and so produce not only a possible aggravation of the symptoms, but a prolongation of the period of subsidence.¹

The Size of the Opening in the Skull.

There is nothing in the cases here brought together that would seem to support Guthrie's belief that hernia cerebri is more likely to occur with a small opening than

¹ 'Brain,' 1893, p. 222.

with a large one. But it must be admitted that a small aperture is more likely to be the result of a concentrated force and to be associated with injuries to the brain and its membranes; whilst in such a wound there is more danger of sources of irritation, such as detached portions of inner table, being left *in situ*.

The Course of Hernia Cerebri.

Hernia cerebri being due to increased bulk consequent upon inflammatory effusion into the brain tissue, will in due time shrink and disappear, as the inflammatory process subsides and the effusion becomes absorbed.

The time required varies, and will depend upon (1) the extent to which the brain is affected, and (2) the absence or presence of continued irritation. It may be a question of a week or two, or possibly of several months. So long, however, as sepsis is avoided this course may be counted upon. But if septic infection should take place, then the outlook is extremely serious. The forms which it may take, viz. meningitis, abscess, and diffuse inflammation, will be discussed elsewhere, but here it may be stated that in abscess only—and then only in a proportion of cases—can there be any hope of saving the patient.

Many of the cases quoted, when of short duration, are probably examples of resolution. Case 10 is evidence of cicatrisation; and of suppuration there are many instances.

Conditions complicating Hernia Cerebri.

1. An occasional consequence is a predisposition to hæmorrhage which takes place into the softened patch, and may cause serious external bleeding. This is, no doubt, due to the vessels in the inflamed area sharing in the general softening and losing the support of the healthy tissue. The onset of hæmorrhage may lead to a sudden increase in size of the protrusion, and Abernethy meeting with two cases of this nature was led to look upon it as

the cause, and not an occasional complication of hernia cerebri.

2. Very sudden enlargement of the tumour may arise from quite trivial causes. Thus, in G—'s case it occurred in connection with a sore throat and a rise of temperature, and in Case 75 it followed an examination of the parts with a probe.

3. The influence of gravity must not be overlooked when the aperture in the skull is low down on the vertex or at the base. If the brain substance were very diffuent the tendency would be for it to pour out, rather than to rise out, of the brain case. The difficulty of managing a protrusion under such circumstances would be enhanced, and an element of danger introduced (Cases 34, 102).

Hernia Cerebri and Abscess.

The prolonged continuance of a large hernia cerebri, or its increase after it has existed for a long time, is nearly always due to some serious condition upon which the protrusion depends. This may be irritation in some form capable of removal (Cases 74, 75) or perhaps beyond the reach of it, but now-a-days, when thorough operations for depressed compound comminuted fractures are the rule, it is more likely to be due to abscess.

It has been stated that a rapid increase in size of a hernia that has been long stationary, or is even shrinking, may result from some slight local or general cause, but an increase of this kind is only transient. The tumour soon returns to its original size.

The formation of a hernia cerebri after a considerable interval has elapsed since the original injury to which it is to be traced, is strongly in favour of abscess (G—'s case).

The question naturally suggests itself why in some cases of brain abscess hernia cerebri is such a marked and persistent feature, whilst in others there is only a slight bulging of the brain into the bony aperture. An explana-

tion is probably to be found in the varying amount and degree of softening which surrounds the abscess or inter-venes between it and the surface. On this point Cases 26 and 27 throw some light.

Prognosis.

So long as sepsis is avoided this is good, but when it is present it may take the form of—

1. *Meningitis*.—A condition always fatal. The infection may be received—

- (a) In connection with the original injury, whether accidental or operative (Cases 21, 22).
- (b) As a consequence of some operative interference with the hernia cerebri (Cases 96, 97).
- (c) From an extension of the inflammation of the brain, which in such cases is, no doubt, of a septic nature.

2. *Abscess*.—This is very serious, but not necessarily fatal.

Recovery may take place—

- (a) By spontaneous evacuation (Cases 17, 18).
- (b) After surgical interference (Cases, G—'s, 78, 79).

Death may result—

- (c) Notwithstanding surgical interference (Case 62).
- (d) From rupture into the lateral ventricle (Cases 93, 94).
- (e) From the supervention of meningitis (Cases 63, 64).

3. *Very extensive disorganisation of the brain*.—Probably most of the cases in which large areas of brain tissue are inflamed are septic, but that this is not always so, a case recorded in 'Brain'¹ by Dr. Conyngham Brown and the writer clearly proves. This case will be referred to more fully, later.

¹ 'Brain,' 1893, p. 213.

Analysis of Cases.

Left to nature—34 cases; 19 recoveries, 15 deaths.

Recoveries—

	Cases.
(a) Receded gradually after formation of hernia cerebri	1—10
(b) Ditto, but dura mater uninjured and gave way from internal pressure	11, 12
(c) Hernia sloughed or separated	13—15
(d) Hernia discharged foreign body and subsided	16
(e) Abscess spontaneously evacuated	17, 18
(f) Insufficiently reported	19

Deaths—

(g) Meningitis	20—23
(h) Meningitis, large area of brain inflamed	24, 25
(j) Abscess	26, 27
(k) Abscess, pyæmia	28
(l) Cause uncertain	29—34

The usual duration of a hernia cerebri, if not subjected to meddlesome surgery, and if recovery takes place, varies between a few days and a couple of months.

Excluding four instances (Cases 18, 19, 35, 37) in which the time is not stated, all the successful cases in this group and the next ran their course within these limits, with the exception of two, in one of which (Case 10) a wedge of cicatricial tissue was found in the brain, some years later, at the site of the protrusion, and in the other (Case 17) the hernia subsided on the evacuation of an abscess. These two cases persisted for four months. Contrast this with the duration of the condition in the third group, in which caustic remedies were used. Only in four of the five recoveries is the time mentioned, but in one (Case 50) the patient was in hospital five months, and in another (Case 51) the hernia existed for double that period.

Sepsis was responsible probably for all the fatal cases, and in the light of modern ideas it is likely enough that had it been possible to use the methods now in vogue, the number of deaths, and possibly the number of cases in which hernia cerebri developed would have been materially diminished.

Case 30, in which a compound depressed fracture was not operated upon for a fortnight, and until symptoms of mischief had made their appearance, is interesting as illustrating the importance of preventive treatment in another direction.

Cases in which Pressure was the chief treatment employed.

Recoveries 11, deaths 4.

<i>Recoveries—</i>	<i>Cases.</i>
(a) Where symptoms were already on the wane when pressure was used	35, 36
(b) Where a source of irritation came away	37
(c) Where pressure seemed to act well	38—40
(d) Where pressure probably influenced recovery but little	41—43
(e) Where a tendency to protrude continued for some time	44, 45
<i>Deaths—</i>	
(f) Abscess	46—48
(g) Uncertain, probably abscess	49

In this group also sepsis was the cause of every death. In one case¹ (47) the result was unavoidable, but in the others early operation and asepsis might have produced different results.

But the eleven successful cases only represent a fraction of those in which pressure was employed. In many others it was tried and given up, but even in those cases in which it was the only active treatment, it was evident that the majority or all of them would have recovered without it.

¹ Fracture of inner table.

Its value, when it had any, probably lay in expediting the natural shrinkage of the tumour, and sometimes, perhaps, in preventing undue growth.

When the hernia has assumed a mushroom shape, its periphery overlapping the edges of the opening in the scalp, pressure is out of place, for it flattens out instead of repressing the growth.

There is, however, evidence to show that the employment of pressure, other than of a very gentle character, is likely to do more harm than good. In Case 11 the pressure exercised by the intact dura mater led to symptoms which were relieved when that membrane gave way and allowed the brain to protrude. In other cases pressure has had to be discontinued because it caused symptoms which were relieved on its removal (Cases 59, 94, 100). But perhaps the most serious result of pressure is the effect it may have in promoting the destruction and absorption of the softened brain tissue, and so favouring some future functional weakness. To illustrate this possible danger, the writer would refer to the case published in 'Brain,'¹ to which allusion has already been made.

Here the right hemisphere had evidently been the seat of a diffuse aseptic inflammation resulting from a fall. Some years afterwards both ventricles were found very much enlarged. The right² was more enlarged than the left,³ and occupied almost the whole hemisphere. It was suggested that the unequal dilatation was the effect of increased intra-ventricular pressure acting upon softened brain upon one side, and normal brain on the other, and no doubt the cicatricial contraction of the inflammatory elements present on the right side had increased the disparity. In the end only a shell of hardened and condensed tissue enclosed the ventricle, and the patient suffered from hemiplegia and frequent convulsive seizures. *Hernia cerebri* has been compared to a safety-valve. In

¹ 'Brain,' 1893, p. 213.

² Fig. 5.

³ Fig. 6.

FIG. 5.

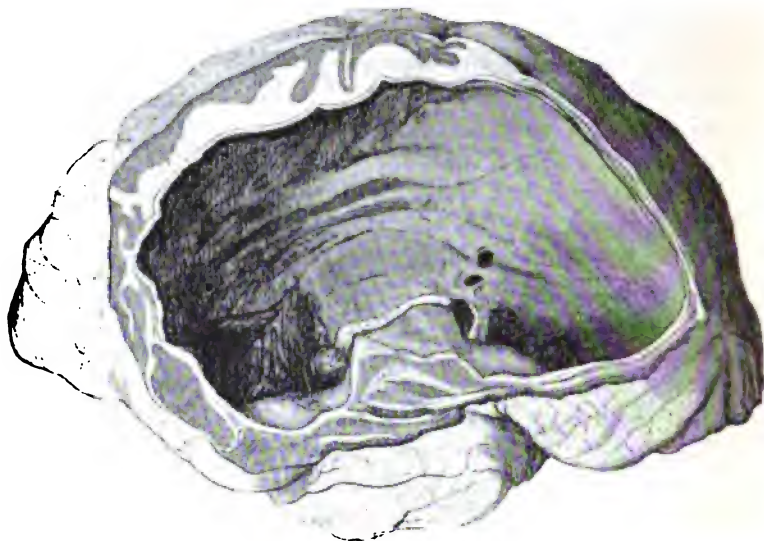
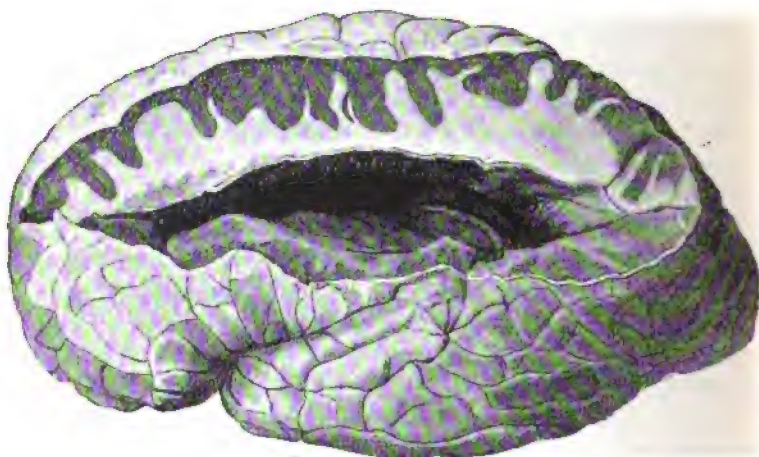


FIG. 6.



From 'Brain.'

the case just narrated the condition was such as would have led to hernia cerebri had there been an aperture in the skull; but how much less grave the subsequent changes would have been, if a large opening had permitted a free expansion of the swollen brain, can only be a matter for speculation.

Cases in which Caustics constituted the chief method of treatment.

Recoveries 5, deaths 2.

Recoveries—

	Cases.
(a) Certainly no benefit	50, 51
(b) Probably no benefit	52, 53
(c) Harm done :	54

In 3 of the above (1 in each division) pressure was combined with caustics.

Deaths—

	Cases.
(d) Meningitis	55
(e) Uncertain	56

Of the deaths, certainly one and probably both were due to sepsis. Caustics were employed in many of the other cases without benefit, but in these, in which they were not followed by other more heroic methods of treatment, and in which recovery ensued, evidence of real benefit is absent. Indeed, in one instance, the patient was saved by the discontinuance of the treatment. Nor is this astonishing, for the frequent destruction of the surface, and the necessary separation of the slough, would be likely to keep the protruded brain in a constant state of irritation, favourable to a continuance of the inflammatory swelling, and to delay in its natural subsidence, at the same time that it would offer increased facilities for septic infection.

The five successful cases, therefore, may be fairly regarded as cures due to natural processes *in spite of* the treatment employed.

Various Operative Procedures—not Removal.

Recoveries 3, deaths 9.

Recoveries—

	Cases.
(a) Transplanting skin flap	57
(b) Puncture by thermo-cautery	58
(c) Surface of hernia cerebri pared off daily, causing bloodletting	59

Deaths—

(d) Extensive laceration of brain, exploration for pus fourth day	60
(e) Large area of brain breaking down, top removed by spatula	61
(f) Abscess, incision, rupture in lateral ven- tricle	62
(g) Meningitis, abscess incised	63, 64
Ditto, subdural abscess opened, later interference with wound	65
Ditto, hernia cerebri removed by patient's fingers ¹	66
Ditto, with pyæmia, laceration of hernia by patient's fingers	67
Ditto, with pyæmia, tumour incised	68

In this group all the recoveries were instances of natural cure, the treatment described being merely unnecessary incidents in the course of the cases. With one exception (Case 60), the fatal cases were due to sepsis, and the suspicion is naturally raised, though with how much justification it is difficult to be sure, that in some the septic infection may have been the result of the operative interference.

Removal of Tumour.

Recoveries 19, deaths 22.

Recoveries—

	Cases.
(a) After removal (5)	69—73
In two (71, 72) pressure subsequently	

¹ This case might perhaps more correctly have been placed in the next division.

Cases.

employed; in one (73) ligature used when tumour had become smaller.

- (b) Source of irritation removed when excision was performed (4) 74—77
 - (i) Bony fragment in centre of tumour, arm remained paralysed (76).
 - (ii) Projecting or loose bone removed (74, 75).
 - (iii) Exit given to intra-cranial discharge (77).
- (c) Excision followed by evacuation or drainage of abscess (2) 78, 79
- (d) Hernia removed more than once (in six several times) (8) 80—87

Deaths—

- (e) Abscess (5) 88—92
 - Ditto, rupture into ventricles (2) 93, 94
- (f) Meningitis (1) 95
 - Ditto, probably induced by operative procedures (2) 96, 97
- (g) Large area of brain disorganised or removed (5) 98—102
- (h) Cause uncertain (7) 103—109

Of the six cases included in groups (b) and (c), one (76) thoroughly justifies the radical treatment employed; two (78, 79) might possibly not have recovered without it as the abscesses presumably were deeply situated; and in two others (74, 75) under present methods no source of irritation would have been left. The other thirteen cases of recovery, groups (a) and (d), would probably have got well if left alone. In seven of them¹ (72, 80, 81, 83,

¹ In Case 72 on the 4th day, in Case 80 on the 12th and 25th days, in Case 81 on the 9th, 23rd, and 42nd days after the injury, in Case 83 several complete or partial removals before the 35th day after the injury, in Case 85 in a few days, and repeated every 2 or 3 days, in Case 86 early removal inferred (it was sliced off daily), in Case 87 on the 19th day, and repeated at intervals for 20 days.

The remarks upon the cases "left to nature" may be compared with these facts.

85, 86, 87) removal was carried out much too soon for nature to have had a chance. In one (73) nature was doing its work, and in another (69) the continued irritation of caustics was probably preventing a natural subsidence. In the remaining four (70, 71, 82, 84) the details are insufficient for special comment, though an early resort to removal may be suspected.

Among the causes of death *cerebral abscess* occurs seven times (88—94). In two (93, 94) rupture into the ventricles was an adequate explanation. In one (89) the discharge of the abscess during life did not prevent a fatal issue.

Meningitis was present in three cases, and probably in all was the essential factor in causing a fatal issue. In two of the three (96, 97), if not in the third, there is a strong suspicion that the removal of the tumour was to blame.

In Cases 98—102 death presumably was due to the *large area of brain involved or removed*. The salient features of this group are the frequency of interference and its early commencement (*vide* Case 100). This continued irritation, whilst increasing the dangers of sepsis, may in some degree account for the large amount of brain implicated. Had cleanliness and non-interference been relied upon, the results might have been more satisfactory.

The cause of death must, for want of definite statements, be put down as *uncertain* in seven cases (103—109), but we can well imagine that sepsis played its part in the majority. Thus in Cases 106 and 107, after active treatment extending over months, the patients succumbed probably to meningitis, for which surgical interference was most likely to blame. In Case 108, said to have died of abscess on the thirty-seventh day, the fungus was three times removed in that short time, and Case 109 lasted little more than a month, but removal of the tumour had led to opening of the lateral ventricle. In two of these cases hæmorrhage was important. Bleeding into and from the tumour led to its removal in Case 104, and in Case 105 the patient nearly bled to death from inability to stop it after excision.

Table to show the Comparison between the Results due to Natural Processes and those following Operation.

<i>Natural processes.</i>					Recovery.	Death.	Total.
(a)	Left to nature	(including G—'s)	20	15	35
(b)	Pressure	11	4	15
(c)	Caustic	5	2	7
					36	21	57
<i>Operation.</i>							
(d)	Various	3	9	12
(e)	Excision	19	22	41
					22	31	53

Summary.

From what has gone before it seems clear that in all the cases in which there was no operative interference, the successful issue was dependent upon natural processes. Pressure and caustics, though freely used, rarely seem to have given any real assistance, and the latter is especially to be condemned.

Then, *of the cases treated by operation*, the three successes under (d) were really instances of natural subsidence, and of the nineteen successes following excision, thirteen at least would have ended in a natural cure if left alone. Of the other six, removal was of value in three, and *perhaps* the only way in which life could have been saved; but in the remainder, though of use, successful results might have been obtained in other ways without the mutilation of so important an organ as the brain—a mutilation which, it can hardly be doubted, caused a permanent paralysis more frequently than is stated.

Sepsis was answerable for the result in all the fatal cases with one or two exceptions, and in connection with this it is perhaps worth while to draw attention to the largely increased percentage of deaths¹ in the operative cases, where opportunities for septic infection must have been largely increased.

¹ The percentage of deaths in cases under heads (a) (b) (c) was 35·8, whilst the percentage in the operative cases was 58·5—an increase of 21·7.

The Treatment of Hernia Cerebri.

1. *Preventive treatment.* (a) *Asepsis.*—As a very large proportion of these cases are due to traumatism, from which septic influences have not been successfully excluded, it is not only reasonable to hope, but it is certain, that very careful attention in rendering all injuries to the scalp and skull as aseptic as possible, must lead to a considerable diminution in the frequency of this condition.

(b) *Thorough treatment of compound depressed fractures of the skull.*—Of the 110 cases in the present paper, 88 owe their origin to compound depressed fractures. The present rule of practice to operate at once upon all cases of this kind is very salutary. The operation should be thorough, all loose fragments being removed. This, further, allows a more thorough cleansing of the wound. It is probably in consequence of the great care that is exercised in these two matters, that hernia cerebri is now so rarely seen, and that so few cases are to be found in recent literature.

2. *Non-interference.*—Except for rigid cleanliness and the careful exclusion of all septic influences, the safest rule of practice is to leave the protrusion to pursue its own course. A careful study of the material upon which this paper is based, brings out this point with great clearness.

3. *Operative treatment.* (a) *When the tumour is associated with abscess.*—Under these circumstances the policy of non-interference must be departed from. Treatment should aim at evacuation and drainage of the abscess—not removal of the tumour, which may contain important motor centres. A method which would entail permanent paralysis should not be entertained without very strong reasons. Spontaneous evacuation is too uncertain to be trusted to, and only likely to occur in those cases where surgical interference would be easy, and, if judicious, almost certainly successful.

(b) *Apart from abscess* the cases in which operative treatment is really justified must be *excessively rare*.

Such a case was 76, where a fragment of bone was found embedded in the base of the tumour. Though in that instance the discovery was only made at the time of the operation, yet in the present day, by the use of the skiagraph, a similar condition could probably be diagnosed, and an attempt would then be made to extract the irritating body. If that were done the necessity for shaving off the protrusion would no longer exist.

ABSTRACTS OF 109 CASES OF HERNIA CEREBRI.

Cases left to Nature ; Recoveries.

CASE 1. Fabrice de Hilden, Spring's paper, 'Mém. Acad. Royale de Méd. de Belge,' iii, p. 70, 1854, Obs. 44.—A young man developed a fungus as a result of fracture of the right parietal. In twenty-four hours it grew as big as a hen's egg. Powders and aromatic lotions were applied. It began to subside in fourteen days, and the wound was perfectly healed in two and a half months.

CASE 2. Hey, 'Practical Observations in Surgery,' p. 12.—Boy aged 14. Compound depressed fracture of right parietal bone, injury to dura mater, and wound of brain. Operation on fourth day. A fungus about the size of a large nutmeg arose from the brain, and had a strong pulsation. No pressure used. In three weeks it had subsided, and the wound then healed.

CASE 3. Hey, 'Prac. Obs. in Surgery,' p. 14.—Boy aged 13. Compound depressed fracture of left parietal bone with lacerated wound of dura. Operation. Through the wounded dura an oblong fungus arose, but with simple dressings without pressure it retired as cicatrisation advanced. The boy got well.

CASE 4. 'Cooper's Surgical Dict.,' p. 691.—The only case Larry ('Mém. de Chir. Mil.,' t. 4, p. 206) ever saw recover was treated by a dressing of slightly camphorated oil of camomile, removing all kinds of irritation, and excluding air. The tumour was small.

CASE 5. Moyle, 'Lond. Med. Report,' vol. viii.—Boy aged 8. Compound depressed fracture of left side of frontal bone, with loss of

brain substance ; compression. Trephined. On the third day hernia cerebri appeared, and increased to size of half an egg. The hernia retired by degrees as the wound cicatrised.

Some years later the boy was in good health.

CASE 6. P. P. Oreagh, 'Med.-Chir. Trans.,' 1811, vol. ii, p. 53.—November 20th.—A wounded seaman had a depressed compound fracture involving chiefly the left parietal bone, the membranes and brain being injured by a splinter. Loose bones removed.

21st.—Brain substance protruding.

25th.—Paresis of one side (not stated). More loose fragments removed.

30th.—Lethargic ; protrusion persists.

December 1st.—Convulsions ; dozing and at times comatose ; paralysis increased. More bone removed, exposing entirely the ruptured membranes, through which the brain protruded.

8th.—Paralysis much improved.

12th.—Protrusion receding.

18th.—Discharge from cerebrum slight and almost serous.

The sores healed and the paralysis recovered.

CASE 7. MacGillivray, 'Australian Med. Journ.,' 1864, vol. ix, p. 163.—Girl aged 3½. Injury over right parietal bone, from which brain protruded on the second day, caused by pickaxe. Marked symptoms. No operative interference. Some bone exfoliated, and in a month hernia cerebri formed as big as a hazel-nut. It was stationary for four weeks, gradually diminished and disappeared, and the wound eventually healed.

CASE 8. Joseph Bell, 'Edin. Med. Journ,' 1868, vol. xiii, p. 1086.—April 13th.—Man aged 25, admitted for compound depressed fracture of right parieto-occipital region with injury of the dura mater, and penetration of brain by a small piece of bone. Loosened fragments were removed.

19th.—Brain protruding through dura mater.

23rd.—Hernia cerebri as large as half an apple.

29th.—Till this date he had had head symptoms, such as delirium. He now began to mend.

May 9th.—Hernia had disappeared, and the wound in dura was apparently closed. Recovery followed.

September 16th.—Left hospital, having been detained so long in consequence of a severe injury to the pelvis, which had complicated the head condition.

CASE 9. Berger, 'Bull. et Mem. de la Soc. de Chir. de Paris,' 1880, vol. vi, p. 253.—Boy, aged 13, came under treatment with a cerebral hernia as large as a hazel-nut, resulting from an untreated injury of the right frontal region four days before. After the removal of some fragments of bone, and liberation of some pus which lay between the dura mater and the brain, the hernia rapidly subsided, and serious symptoms disappeared. Cure resulted, but the mental condition remained impaired.

CASE 10. John Duncan, 'Brain,' 1879-80, vol. ii, p. 413.—Boy aged 13. 1st day.—Pony kick caused compound depressed fracture of left frontal bone; portions of bone removed.

11th.—Hernia cerebri developed without symptoms.

21st.—As large as a small orange. For four months it remained stationary, only cleanliness being observed, then it dwindled.

6th month.—A level pulsating scar remained.

After-history.—Three years later he died of inherited phthisis.

Post-mortem.—A circular aperture 1 inch in diameter was found in the bone, and from this, as a base, an irregular cone of cicatricial tissue extended into the brain for $\frac{3}{4}$ inch. It occupied the centre of the second and part of the first convolution.

CASE 11. Joseph Taylor, recorded by Stanley, 'Med.-Chir. Trans.,' vol. viii, p. 39.—Boy aged 10. 1st day.—Compound depressed fracture of left parietal bone.

3rd.—Trephined; compression symptoms continued.

7th.—Right facial paresis.

8th.—Tongue paralytic; curious brain symptoms; involuntary micturition. Improved in the evening; became compos; slept; paralysis less perceptible.

9th.—On removing dressings the dura was found to have ruptured, and natural and apparently healthy brain protruded to the size of a moderate hen's egg. Untoward symptoms had disappeared. Compression of the hernia caused restlessness and stupidity.

Wound healed rapidly.

24th.—Boy playing at marbles with his companions.

CASE 12. Spanton, 'Lancet,' 1864, vol. i, p. 268.—August 6th.—Boy aged 6. Compound depressed fracture of left side of frontal bone from a horse kick; compression. Operation by Mr. Favell; dura mater uninjured.

26th.—Progressed favorably, when to-day the dura mater gave way and brain protruded: free discharge of pus. The hernia reached

the size of half a walnut, and was painted every other day with collodion.

October 22nd.—Surface of the wound level.

November and December.—Portions of necrosed bone came away.

December 31st.—Healed completely.

CASE 13. G. Lowe, 'Lancet,' 1850, vol. i, p. 759.—A groom had a compound comminuted fracture of the skull with laceration of brain from a horse's kick. A teaspoonful of brain substance was lost in removing a deeply embedded piece of bone.

Inflammation of the brain came on with drowsiness; great slowness of reply and sickness; and in ten days a hernia cerebri formed, which at last attained the size of a walnut. On the fourteenth day, under mercury, symptoms abated; the tumour shrank considerably, and finally sloughed away. Recovery was perfect.

CASE 14. From medical commentaries, Stanley, 'Med.-Chir. Trans.' vol. viii, p. 38.—Protrusion of brain began on the fourth day, and increased gradually till the fourteenth, when it spontaneously dropped off in pretty large pieces. Only treatment dry lint dressing.

CASE 15. Lapeyronie, Spring's paper, Obs. 51.—In this case a fungus followed the operation of trephining for injury. The dura mater had been wounded. Small portions of brain (nut) came away at each dressing for ten days. A cure of the hernia resulted. No treatment mentioned.

CASE 16. G. B. Mallet, 'Trans. Prov. Med. and Surg. Assoc.' vol. vii, p. 343.—J. B—, aged 18. Compound depressed fracture of vertex. Bone driven into brain removed. In eight days a growth from the brain appeared, persisted a week, discharged a piece of coal as large as a pea, and then gradually disappeared. No ill effects left.

CASE 17. W. J. Goodeve, 'Lond. Med. and Phys. Journ.,' 1823, vol. 50, p. 191.—1st day.—Man aged 30. Scalp wound caused by falling stone, followed by symptoms.

6th.—Trephined; dura mater found puffed up; opened. A quantity of serum seen collected under arachnoid.

8th.—Rigors; right hemiplegia becoming almost complete.

12th.—Hernia cerebri.

35th.—Hernia 2 inches in diameter and 2 inches in height.

46th.—Smaller from sloughing of its surface.

48th.—Sloughing continues.

50th.—Bloody serous discharge.

60th.—Sloughing and bloody serous discharge continue.

69th.—A quantity of brain-like substance has found its way through a hole in the centre of the tumour. A probe passes into what was apparently an abscess cavity.

73rd.—Suppuration free; tumour lessening.

96th.—Continued shrinking; hardly any suppuration.

117th.—No vestige of tumour; wound healed.

After-result.—Epileptic fits at first, but the tendency diminished. When reported he had had none for twelve months, and was doing his work as a farm labourer. Paralysis evidently better.

CASE 18. Elcan, 'Amer. Journ. Med. Sci.,' April, 1880; Starr, 'Brain Surgery,' p. 183.—A boy suffered from a compound comminuted fracture of the left frontal bone, from which brain matter was expressed. In a few days hemiplegia and aphasia developed, and the boy became comatose.

Operation.—Bone elevated and portions removed. Hernia cerebri ensued; aphasia and hemiplegia remained, but consciousness was clear. Four days later the wound was again examined, and during its manipulation eight ounces of pus were suddenly evacuated from an abscess which unexpectedly broke. Subsequently the aphasia and paralysis subsided, the wound healed, and the boy recovered.

CASE 19. Ambroise Paré, Spring's paper, 'Mém. Acad. Royale de Méd. de Belge,' vol. iii, p. 70, 1854, Obs. 43.—A French seigneur, wounded at the siege of Metz in 1552, developed a fungus which came from the dura mater. It increased daily. The patient was perfectly cured.

Cases left to Nature; Deaths.

CASE 20. Marshall, 'Med. Times and Gazette,' 1875, vol. ii, p. 679.—February 23rd.—Man aged 14. Depressed compound fracture of left parietal bone with loss of brain substance from falling on a spike; trephined.

25th.—Temp. 103°; hernia cerebri formed; increased considerably.

26th.—Death.

Post-mortem.—Meningitis.

CASE 21. 'London Med. Gaz.,' 1829, vol. iv, p. 444.—A man aged 48 was operated on in the Glasgow Royal Infirmary for depressed

compound fracture of the left parietal bone, and the dura was found wounded. He died on the sixth day of meningitis, just as a fungus was making its appearance. The wound was sloughy.

The *fungus* was the size of a pigeon's egg. It was of a dark brown colour, gradually turning to grey as it passed into the surrounding cerebral substance. Its central part was soft, and immediately around it the brain presented numerous bloody points; elsewhere the brain was firm.

CASE 22. Abernethy, 'Surgical Observations,' p. 51.—Man aged 40 received a depressed fracture of the parietal bone from a falling stone. Operation. Delirium, followed by increasing coma, ensued. On tenth day a hernia cerebri rose through an ulcerated opening in dura mater; increased to size of a hen's egg. Death on twelfth day.

Post-mortem.—The tumour was larger than before, and of a dark colour. Hæmorrhage had taken place from the hernia into the dressings to a considerable extent. The tumour appeared to consist of coagulated blood of a fibrous texture, and to have originated within the substance of the brain about an inch below the surface. The pia mater was inflamed.

CASE 23. Abernethy, 'Surgical Observations,' "Injuries of the Head," p. 54.—A carpenter, in addition to other injuries, received a depressed compound fracture of the right parietal bone. The bone was trephined and elevated. Twelve days after the accident a hernia cerebri appeared rising through an aperture in the dura. It increased rapidly, and two days later he died.

Post-mortem.—Meningitis. The hernia cerebri was formed of congealed blood deposited in the medullary part of the cerebrum, the containing cavity being an inch in diameter, and its parietes appearing to be the substance of the brain condensed by pressure. The ventricles were full of a serous fluid mixed with blood.

CASE 24. Bouchacourt, 'Bulletins de la Soc. Anat. de Paris,' 1838, vol. xiii, p. 13.—Man aged 32 had a severe fracture of the anterior part of the skull with injury to brain. On the sixth day inflammatory symptoms came on, and a cerebral hernia which had formed became inflamed and gangrenous, and separated in portions. On the granulating surface which formed, a fistulous track opened and discharged limpid fluid in drops. He began to go wrong on the twenty-fifth day, and died on the thirty-second of meningitis.

Post-mortem.—A short fistulous track traversed soft diffuent brain, which intervened between the surface of the wound, largely cicatrised, and the lateral ventricle.

CASE 25. G. W. Callender, 'Med.-Chir. Trans.,' vol. liv, 1871, p. 132.—December, 1863.—Sailor aged 27. Fracture of skull. A month later trephined and pus liberated. Insensibility two months; loss of sight, followed by convulsive seizures and paresis of left limbs.

1864.—Removal of necrosed right parietal bone.

1866.—After an injury, complete paralysis of left side. Fits ceased.

1870.—Fell on his head when drunk. Next morning found a swelling on right side of skull, which steadily increased. Admitted into St. Bartholomew's a week later with hernia cerebri as large as a small orange. Had rigors; became noisy, then insensible, and died nine days after admission. Left-sided paralysis had persisted. Left eye blind. Right eye slight vision.

Post-mortem.—Evidence of long past meningitis. Softening of considerable portion of right hemisphere from which hernia cerebri protruded. The softening in places amounted to fluidity of brain tissue. Recent meningitis.

CASE 26. Norris, 'Amer. Journ. Med. Sci.' 1838-9, vol. xxiii, p. 279.—July 13th.—Woman aged 37. Trephined for depressed compound fracture of right temporo-parietal area. Dura slightly wounded. Paralysis of left arm and then left leg developed with other head symptoms.

August 1st.—Hernia cerebri.

16th.—Hernia as large as an egg.

19th.—Sloughing commencing at its top.

21st.—Lingering death.

Post-mortem.—Tumour arose from cerebrum, which it resembled in consistence and appearance. A large deep abscess existed in the cerebrum at the side of the tumour, and nearly the whole of that side of the brain was disorganised.

CASE 27. Norris, 'Amer. Journ. Med. Sci.,' vol. xxv, p. 269.—On July 1st a boy aged 16 received a compound depressed fracture of the left side of the frontal bone an inch above the orbital ridge, an iron bolt being fixed in it. On its removal, and also some loose portions of bone, a portion of lacerated brain as big as a walnut protruded. There was right arm paralysis. The protrusion subsided until there was only a slight rising above the surface of the bone, and on the 19th the paralysis was entirely gone. Cicatrisation went on slowly till August 18th, when untoward symptoms developed, and he died on August 30th.

Post-mortem.—A fragment of in-driven bone was pressing on the

brain. An abscess occupied the whole anterior lobe. It contained 5 or 6 ounces of pus. The walls were lined by a thick false membrane. The cerebral matter around it was of a light yellowish colour and softened. Beneath the opening in the skull the dura was adherent to the brain, and the abscess was immediately beneath it. Ventricles much distended with serum. Right hemisphere normal.

CASE 28. Ward, 'Lancet,' 1848, vol. i, p. 305.—1st day.—Boy aged 7. Quoit accident. Compound fracture of left side of frontal bone. Depressed fragments removed. Superficial laceration of dura.

9th day.—Hernia cerebri formed.

12th to 23rd.—Pyæmic symptoms. Death.

Post-mortem.—At the base of the protrusion¹ was a small abscess. Several purulent deposits in the lung.

CASE 29. James Syme, 'Edin. Med. Journ.,' 1833, vol. xxxix, p. 315.—Man aged 28 received a depressed compound fracture of the right parietal bone with loss of brain substance. The depressed bone was removed. Paralysis of left arm and leg was followed by coma and death on the fourth day.

Post-mortem.—A cerebral hernia as large as a walnut. The convolutions of the subjacent brain, expanded, softened, and injected with blood, could be distinctly traced into the tumour, the principal part of which was composed of coagulated blood. The cerebral substance in the immediate neighbourhood of the injury was reduced to a pulpy consistence and tinged of a blood-red colour. In other parts nothing remarkable.

CASE 30. Carmichael, 'Dublin Med. Press,' 1841, vol. v, p. 179.—A man with a compound depressed fracture of the skull was not operated on for a fortnight when symptoms had appeared. Loose portions of bone were removed. The same stupid, listless state continued, and a few days later a fungus began to protrude through the opening. This was left alone, and in eight or ten days it declined, and finally disappeared. Death five weeks after the injury.

Post-mortem.—Where the fungus had existed there was a large slough, and the neighbouring parts of the brain were in a softened state.

¹ This was supposed to be connected only with dura mater, the protrusion not containing cerebral structure.

CASE 31. Thorndike, 'Boston Med. and Surg. Journ.,' 1877, p. 82.—Man aged 32. Extensive compound depressed fracture of right parietal region with laceration of brain and symptoms of compression, the result of a fall. Loose bone removed, leaving opening 3 inches by $2\frac{1}{2}$ inches. Improvement followed. In a week palsy of left hand. This had been predicted because temperature had been 102° in the left axilla and 99.9° in the right, and this fact had been noted in other cases of palsy following head injury. In three weeks hernia cerebri developed, and grew to 4 inches in diameter. Palsy affected all the left side. Death from exhaustion eighty days from the accident. He became unconscious before death. No autopsy.

CASE 32. Abernethy, 'Surgical Observations,' p. 38.—Boy aged 14 was trephined for depressed fracture of the anterior inferior angle of parietal and part of frontal bones. Middle meningeal hæmorrhage. On removal of clot the depressed dura mater rose to its original level, and bleeding ceased. He recovered consciousness towards the close of the operation. After some symptoms on the fifteenth day he had rigors and pain in the head, and next day a hernia cerebri was found protruding through the dura. It increased to the size of an orange in twenty-four hours. Death next morning. No post-mortem.

CASE 33. G. Lowe, 'Lancet,' 1850, vol. i, p. 759.—Man aged 19. Extensive compound depressed fracture of skull from falling brick. Brain lacerated, and some escaped. Operation.

On eighth day symptoms of inflammation of brain became threatening, and a fungous growth appeared. Calomel freely given; inflammation of brain subsided, fungus sloughed away, leaving healthy granulating surface. In fourth week headache and fever, and reappearance of hernia; then coma and death a month from the accident.

CASE 34. Cæsar Hawkins, 'Lond. Med. Gaz.,' 1832, vol. x, p. 251.—Boy aged 11. Accidental shot, injury of face below the right eye. The middle fossa injured, and brain protruded into the cavity made by the bullet. On the seventh day it had protruded from the face wound to the size of a walnut along a track four inches long. Death on the eighth day.

Post-mortem.—The protrusion was in the usual soft and pulpy state of fungus of the brain, and the cerebrum around the softened part was vascular and of the yellow colour generally found in such cases.

*Cases in which Compression was the chief Treatment ;
Recoveries.*

CASE 35. Bryant, 'New England Med. and Surg. Journal,' 1813, vol. ii, p. 280.—Hernia cerebri formed on the fourth day after the operation of trephining for fractured skull. It increased to the height of an inch, suppurating freely. Being lessened to half its size by suppuration, pressure was applied, and in a few days reduced it wholly, and the boy soon recovered.

CASE 36. R. M. Craven, 'Brit. Med. Journ.,' 1865, vol. ii, p. 522 (reported by T. M. Evans).—March 1st.—Boy aged 15 was operated upon for depressed compound fracture of left parietal bone close to median line, with escape of brain substance.

2nd.—Complete paralysis of right arm and leg without anæsthesia and facial paresis.

3rd.—Right facial paralysis.

5th.—Considerable hernia cerebri.

12th.—Size of half an orange.

21st.—Paralysis of face less.

28th.—Slight pressure applied.

April.—Diminution of hernia; paralysis improved.

May 2nd.—Hernia greatly reduced.

10th.—None left.

July 4th.—Wound healed; able to use arm, and walk.

CASE 37. G. B. Mallet, 'Trans. Provincial Med. and Surg. Assoc.,' vol. vii, p. 343.—J. I—, aged 36. Portions of bone and coal which had penetrated the brain were removed from a compound depressed fracture of the frontal bone. Hernia cerebri on the tenth day. It reached the size of a walnut, and remained stationary till small portions of the inner table came away; then, assisted by slight pressure, it gradually disappeared, and the man recovered. Death from apoplexy five or six years later.

CASE 38. J. W. Heustis, 'Amer. Journ. Med. Sci.,' 1828 or 1829.—Girl aged 10. Fungus followed a severe compound fracture of the skull, with escape of brain matter. He confined it with a strip of adhesive plaster over the protrusion. In a few days it disappeared, and the child recovered.

CASE 39. Bedford Brown, 'Amer. Journ. Med. Sci.,' New Series, vol. xl, p. 399.—Boy aged 10 was operated upon for a kick on the right side of the frontal bone. A large portion of bone was removed, lacerated dura mater cut away, and the brain found lacerated, and portions separated. Inflammatory symptoms in thirty-six hours, subsided in three or four days. On the eighth day a large fungus cerebri opened up the suppurating wound and filled the opening (2 inches \times $1\frac{1}{2}$ inches), and rose 1 inch above the bone. Sponge pressure was used, and in a few days the hernia had subsided, and in three or four weeks the wound was healed. Eight months later the patient was in good health.

CASE 40. C. K. Crawford, 'Edin. Med. and Surg. Journ.,' vol. xii, 1816.—June 25th, 1813.—Boy aged 2 to 3 years trephined for depressed fracture of left parietal bone with loss of brain substance. Hernia cerebri beginning on fourth or fifth day grew rapidly to size of pullet's egg, and was increasing daily. By carefully applied pressure the tumour was much diminished in a few days, and receded very fast, the granulating surface eventually skinning over.

November, 1813.—Seen, and quite well.

CASE 41. Moyle, 'Lond. Med. Reposit.,' vol. viii.—Boy aged 10. March 15th.—Simple comminuted depressed fracture of left parietal bone. Symptoms of compression. Operation. Membranes penetrated by portion of bone, and brain matter discharged for three or four days.

22nd.—Fungus tumour appeared through the wounded meninges, and increased in two days to size of pullet's egg. It could be replaced by finger pressure. It was treated successfully by compression, carried out for several weeks.

July 21st.—Well.

For twenty-three days after the accident he was senseless, with general paralysis of the whole body. Then his senses returned, and paralysis gradually disappeared. No convulsions at any time, and twenty-nine years later he was alive, and suffering no inconvenience from the brain injury.

CASE 42. R. W. Oughton, 'Lancet,' 1850, vol. i, p. 405.—September 4th.—Man aged 30 received a severe compound fracture of the frontal bone, which was accompanied by slight protrusion of brain substance.

5th.—Considerable cerebral protrusion.

6th.—During the next fortnight repeated protrusion, followed by sloughing, occurred. Then gentle pressure by a broad piece of

strapping applied, with gradually increasing tightness, and by the end of the month the hernia was replaced by healthy granulations.

October 15th.—Healing; no tendency to protrusion.

Patient recovered.

CASE 43. Meldon, 'Dubl. Journ. Med. Sci.,' 1872, vol. liii, p. 295.
—Boy aged 7. Double depressed fracture of skull in parietal and occipital regions; dura injured in the parietal wound. Bone elevated, and fragments removed. Three days later rigors and pain. 12th day.—Hernia cerebri appeared, and grew to size of a pigeon's egg. Pressure by lint and tea lead was used. Too much pressure caused convulsive movements. In two or three weeks it had entirely disappeared, and the wound closed. Complete recovery.

CASE 44. Weir, 'N. York Journ. of Med.,' 3rd ser., vol. vii, p. 291 (Case 3 from 'N. York Hosp. Records').—September 12th.—Boy aged 15. Compound depressed fracture of left side of frontal bone. Trephined. Two tablespoonfuls of brain matter escaped.

20th.—Hernia cerebri. Pressure employed, causing no symptoms.

27th.—Tendency to protrude ceased about this date.

November 17th.—Wound healed. Discharged cured.

CASE 45. A. G. Creagh, 'Lancet,' Feb. 21st, 1891, vol. i, p. 423.—June 20th.—Hottentot boy. Fragments of bone removed from a compound fracture of the left parietal bone. The dura was extensively injured, and cortical substance protruded.

24th.—Paralysis of right arm, hand, and leg. Brain injury was situated over the motor area. Patient dull, apathetic; difficulty in expressing his ideas.

26th.—Convulsion.

29th.—Some sloughy brain substance removed from wound.

July 1st.—Hernia cerebri (walnut). Bandage pressure.

3rd.—Tongue protruded to right side.

16th.—Hernia size of fowl's egg. Paralysis complete.

August 26th.—Under continuous pressure the tumour has become level with the scalp.

September 1st.—The wound is skinning over. Gain in power in arm and leg.

October 30th.—Can walk with a crutch, and arm considerably improved.

Cases treated by Compression; Deaths.

CASE 46. Weir, 'N. York Journ. of Med.,' 3rd ser., vol. vii, p. 291 (Case 4 from 'N. York Hosp. Records').—May 29th.—Man aged 24 received a compound depressed fracture of right parietal bone.

June 1st.—Trephined. Dura mater found lacerated, and brain protruded.

8th.—Protuberance in wound.

12th.—Convulsions.

16th.—Hernia reappeared from bandages slipping off; reduced by fingers, some pus and softened brain escaping.

17th.—Coma.

18th.—Death.

Post-mortem.—Purulent material was found around the receded tumour, and beneath it an abscess capable of lodging the last phalanx of the thumb. The brain substance around was softened. Both ventricles moderately distended with fluid. In other parts the brain was normal.

CASE 47. Cabot, 'Boston Med. and Surg. Journ.,' vol. lv, 227, 1856.—May 9th.—Man aged 25. Injury over right parietal bone.

31st.—Left-sided paresis and head pain.

June 3rd.—Operation. Portion of inner table had been detached. Pus evacuated from between dura mater and bone. Serious head symptoms continued during June.

July 4th.—Offensive slough removed.

5th, 6th.—Hernia cerebri. Scalp yielding to let it pass through.

7th.—As large as a hen's egg. Gave feeling of fluctuation.

8th.—It came off spontaneously. An abscess containing an ounce or more of thick bloody pus was found in its interior. Hernia cerebri continued to form in spite of sponge pressure. Portions died and were cut away or separated.

25th.—Death.

Post-mortem.—A large abscess cavity (diameter $1\frac{1}{2}$ to 2 inches) containing small quantity of pus was found beneath the hernia. Its walls were pretty firm. The greater portion of the remainder of the hemisphere was markedly softened. There were numerous hæmorrhages. The right crus was softened and swollen. The left hemisphere was normal.

CASE 48. Harvey, 'Lancet, 1846, ii, p. 503.—June 30th.—Man aged 30, kicked by a horse. Compound depressed fracture of anterior inferior angle of left parietal bone with escape of brain substance.

July 5th.—Symptoms led to trephining.

6th—19th.—Hernia cerebri (chestnut). Stationary. Superficial sloughing.

20th.—Hernia considerably increased. Speech more defective. Paresis of right arm and probably leg; pain.

20th—23rd.—Hernia increasing. Convulsions.

23rd—27th.—Hernia stationary. Speech and paresis improving. Separation of slough.

28th.—Pressure applied.

August 1st.—Hernia rather smaller, measures $2\frac{1}{2}$ inches by $1\frac{1}{2}$ inches by $\frac{1}{2}$ inch in height.

7th.—Nearly level with scalp.

11th—13th.—Swelling formed above zygoma; thought to be protrusion of brain under skin. Slight increase of paralysis. Exfoliation of portions of dead bone. Skinning of wound advancing.

17th.—Rigor, coma, dilated pupils.

18th.—Sensible. Hernia increased, also paralysis.

23rd.—Downstairs. Arm useless. Protrusion receded to level of scalp.

25th—30th.—Paralysis improving. Enlargement of temporal region diminished.

October 2nd.—Vomiting. Hernia protruding again considerably. Very tense. Pieces of loose bone removed.

8th.—Hernia shrunk to level of scalp, but condition not satisfactory.

9th.—Death.

Post-mortem.—Hernia had sunk below scalp level, and the temporal swelling was entirely gone. An abscess containing a small fragment of bone and 2 ounces of pus was found in the left middle lobe. The overlying brain substance corresponding to the seat of injury was much softer than that adjacent to it. The abscess was surrounded by a hardened area.

CASE 49. Moyle, 'Lond. Med. Report,' vol. viii.—A miner had a considerable portion of the left parietal bone driven into the brain. It was removed. Hernia cerebri of considerable size formed in a few days, and was treated by gentle pressure. A sloughing came on, and in six weeks the wound was completely healed. Soon after the right side became completely paralysed, and eight weeks later he was seized with a violent pain in the head, and died in a short time. No post-mortem.

Cases treated by Caustics ; Recoveries.

CASE 50. J. K. Sampson, 'Med. Times and Gaz.,' 1858, vol. ii, p. 571.—November 5th, 1857.—Boy aged 12 admitted with depressed compound fracture of left side of frontal bone with laceration of membranes and brain. Symptoms occurred in four days as granulation advanced and loose bones came away. Hernia cerebri protruded until it overlapped the entire wound. It was treated by repeated dustings of dried sulphate of zinc and moderate pressure, and once when the dressings were left off for three hours the protrusion nearly reached its original size.

April 6th, 1858.—Left the hospital well.

CASE 51. Volcher Koyter, Spring's paper, Obs. 47.—An excrescence formed at the third month in a man whose frontal bone and brain had been injured. It was treated with caustic lotions and other applications, but grew steadily. Towards the end of the eleventh month it ceased to reproduce itself. Cure resulted at the end of thirteen months.

CASE 52. Weir, 'New York Journ. of Med.,' 3rd ser., vol. vii, p. 291 (Case 45, 'Hospital Records').—December 5th.—Man aged 24. Compound depressed fracture of left fronto-parietal region.

December 12th.—Trephined for symptoms of irritation and compression.

14th.—Hernia cerebri.

26th.—Has enlarged a little. Treatment: nitrate of silver and moderate pressure.

January 1st—10th.—Hernia decreasing slowly.

March 7th.—Discharged cured.

CASE 53. Weir, 'New York Journ. of Med.,' 3rd ser., vol. vii, p. 291 (Case 32 from 'New York Hospital Records').—October 26th.—Female aged 22. Compound fracture of left parietal produced by hatchet, with wound of dura mater and brain, and paralysis of right arm.

27th.—Operation.

November 6th—23th.—Protrusion about the size of a hickory nut. Nitrate of silver daily applied.

December 9th.—Reduced to level of integument.

27th.—Discharged cured. No paralysis remaining.

CASE 54. Armour, 'Glasgow Med. Journ.,' 1831, vol. iv, p. 341.—A man after operations for extensive depressed fracture of the skull suffered from a cerebral hernia which was treated by pressure and burnt alum. Progress was bad, and at last the surgeon said he was to be left alone, as he was dying. He was then insensible. Next morning he was much better. Nature's hint was taken and only simple dressings applied, and he eventually left the hospital well, but with somewhat weakened mind.

Cases treated by Caustics ; Deaths.

CASE 55. Emmanuel Koenig de Bâle, Spring's paper, Obs. 48.—A woman aged 40 developed a tumour, thought to be brain substance, after operative procedures for disease over the left temple. This increased daily; then the patient became drowsy, and died. It is to be inferred from Spring's paper that caustics were employed here. Death was due to meningitis.

CASE 56. Moyle, 'Lond. Med. Reposit.,' vol. viii.—March 23rd.—Boy aged 11. Depressed fracture of right parietal bone. Elevation and removal of loose pieces; large aperture in membranes, and escape of brain.

April 2nd.—Left-sided convulsions. Fungous tumour appeared, and in a few days grew to a considerable size. Coma present subsequently. Caustics and pressure of no use. He lingered with frequent and violent convulsions.

28th.—He expired in a fit.

Cases treated by Various Operative Procedures, not Removal ; Recoveries.

CASE 57. J. E. Adams, 'Lancet,' 1876, vol. ii, p. 679.—Jew girl aged 7. Three months after a compound comminuted fracture of right fronto-temporal region with escape of brain substance, had a granulating pulsating mass as big as half a hen's egg projecting above the level of the skin. Pressure only flattened it out. It was covered by transplanting a flap of scalp on to it.

When reported, the size of the protrusion had much diminished, and no pulsation could be felt through the skin. The injury had been received in the early part of the year.

CASE 58. Folet, 'Lancet,' 1890, vol. ii, p. 669, Sept. 27th (Lawson refers to a case under Folet).—Hernia cerebri came on very rapidly, and was punctured in several places with the thermo-cautery, but without improvement. "The protruding mass, which consisted of granulation tissue, only gradually subsided, and cicatrised at the end of five months."

CASE 59. Spaulding, 'New England Journ. Med. and Surg.,' vol. ix, p. 19, 1820.—A young man was trephined for compound depressed fracture of the frontal bone with injury to the membranes. Inflammation supervened, and on the fifth day the integuments began to rise, and the brain burst out, with coincident improvement in the patient's symptoms. If a compressive bandage was drawn too tight it caused violent twitchings of the whole body. The surface of the part protruded was daily pared off, with the loss of about a tablespoonful of blood. The hernia gradually receded within the skull, leaving a depression behind.

Spaulding attributed the good result to local depletion, and did not favour excision.

*Cases treated by Various Operative Procedures, not
Removal; Deaths.*

CASE 60. Weir, 'New York Journ. of Med.,' 3rd ser., vol. vii, p. 291 (Case 2, 'Hosp. Records').—March 30th.—Man aged 36. Compound depressed fracture of left parietal bone with laceration of dura mater and brain. Inability to speak; conscious; trephined.

April 1st.—Comatose; hernia cerebri protruding in a softened state, and easily detached. Female catheter introduced nearly two inches into brain, almost without resistance. No pus reached.

2nd.—Death.

Post-mortem.—The substance of the left hemisphere was broken up to the depth of $1\frac{1}{2}$ inches below the fracture. No pus was seen.

The injury had been caused by a blow from a blacksmith's hammer.

CASE 61. T. G. Coombe, 'Lond. Med. and Phys. Journ.,' 1816, vol. xxxvi, p. 449.—Boy aged 15. Operation for compound depressed fracture of frontal and left parietal bones with laceration of dura mater, followed by relief of compression symptoms. On tenth day hernia cerebri covered by pia mater burst through the healing wound. Compression symptoms returned, and increased with the

growth of the tumour. Its unhealthy top was removed by a spatula, and a thin bloody fluid discharged for twenty-four hours to some ounces, and relieved the symptoms, sensibility returning. He had no return of these symptoms, but sank very fast, and died on thirtieth day.

Post-mortem.—A third of the left hemisphere at its anterior part was converted into a thick dark-coloured fluid, which appeared to consist of blood with brain in a state of suppuration.

CASE 62. Detmold, 'Amer. Journ.,' 1830, Spring's paper, Obs. 56.—Three ounces of pus were let out from an abscess in the left anterior lobe of the brain. Hernia cerebri ten days later. Compression seemed satisfactory at first. Three weeks after operation loss of memory, aphasia, and other symptoms appeared, and hernia tended to return. Another incision failed to find pus, but symptoms were ameliorated, and the hernia subsided. Five days later it reappeared with shivering and coma. A fresh incision in the neighbourhood of the lateral ventricle let out three ounces of pus. Death in two hours.

Post-mortem.—Brain shrunk, normal aspect, and without alteration in its consistency. Both ventricles were full of very liquid pus. Purulent lymph covered the choroid plexuses.

CASE 63. Baudin, 'Gaz. Méd. de Paris,' 1840, p. 271.—A child aged 6 was run over and trodden on, receiving a compound fracture of the right temporal and parietal bone with loss of brain substance, on August 22nd. An abscess containing pus and shreds of brain matter opened on September 1st. Erysipelas followed, and the child died on September 11th.

Post-mortem.—A hernia cerebri was found to have passed through a gap in the centre of the depressed fracture. Meningitis.

CASE 64. Cabot, 'Boston Med. and Surg. Journ.,' lxi, p. 181.—Child aged 3½ years. Cerebral hernia occurred seven days after removal of bone from right frontal region for severe compound fracture with injury to brain. It increased in spite of sponge pressure. On twentieth day, fluctuation being present, an opening was made, and half an ounce of thin watery pus escaped. A fluid discharged constantly from the opening in the hernia, and on analysis was found indistinguishable from other serous fluids. Death on twenty-eighth day.

Post-mortem.—Meningitis. Nearly the whole of the anterior half of the right hemisphere, with the exception of the corpus striatum, was much softened.

CASE 65. Beevor and Horsley, 'Ophthal. Soc. Trans.,' vol. xii (1892), p. 204.—Christmas, 1890.—T. G. L—, aged 13, was trodden on the left side of his head by a horse. Symptoms pointing to intracranial mischief gradually came on and increased.

March 9th, 1891.—Admitted. Double optic neuritis; right hemianopia; drowsy and stupid. Adherent scar above external auditory meatus. Temperature subnormal. Pain.

13th.—*Operation*.—Fracture and portion of necrosed bone found. Considerable area of bone removed. Dura opened, pus found. Softened brain protruded (angular gyrus). Drainage-tube. Flap replaced.

24th.—Marked hernia cerebri. Wounds draining well.

April 22nd.—Mental condition improving.

May 12th.—Optic neuritis subsiding.

19th.—Evening temperature 103°. Wound tense; drowsy last three or four days. Explored; no definite collection of pus found. Wound freely opened. Extra drainage-tube inserted.

22nd.—Unconscious. Cheyne-Stokes respiration. Death in five hours.

Post-mortem.—Hernia involved parts of supra-marginal and angular gyri, &c. Abscess cavity obliterated. Membrane adherent to surface of brain. Softening extended some distance into the brain adjacent to the protrusion. Basal suppurative meningitis.

CASE 66. W. B. Husband, 'Assoc. Med. Journ.,' 1856, p. 355.—February 29th.—Man aged 25 received a compound fracture of anterior superior angle of left parietal bone, with injury to brain, from a horse kick.

March 9th.—Fætor of discharge.

11th.—Drowsiness. Right hemiplegia. Portions of loose bone removed by operation.

12th.—Aphasic symptoms. Hernia cerebri commencing.

13th—14th.—Increasing.

15th.—Looks sloughy.

16th.—Removed by patient's fingers.

27th.—Hernia cerebri size of pullet's egg; has sloughed; ready to be detached; removed by forceps.

April 5th.—Gradually got weaker without much alteration in his mental condition (aphasia, paralysis). Death somewhat suddenly.

Post-mortem.—Meningitis. Brain corresponding to aperture in bone dark coloured and sloughy, but only superficially. Right hemisphere healthy. No mark of abscess in cerebral substance other than in immediate contiguity to the injured part.

CASE 67. Stanley, 'Lond. Med. Gaz.,' 1846, vol. ii, p. 1098.—1st day.—Youth aged 17. Fall against a cart-axle produced an extensive depressed compound fracture of frontal and parietal bones on left side. Brain substance protruded. Other injury to bone from a horse kick. Depressed bone raised, and parts removed.

6th.—Recovery of consciousness. Continued to do well. A hernia cerebri formed and granulated, forming part of the surface of the wound.

19th.—Symptoms of inflammation of membranes; marked left facial paralysis.

25th.—Patient tore dressing off, and lacerated protruded brain with his fingers. Pulse became slow. Coma set in. Convulsions of right side of face.

33rd.—Death.

Post-mortem.—The left cerebral hemisphere was extensively disorganised opposite the external wound, the disorganisation penetrating nearly to the lateral ventricle. The protruded brain was black and sloughy. Meningitis especially on left side and at the base. Fracture of the petrous bone might have caused facial paralysis. Abscesses in lungs.

CASE 68. Ward, 'Lancet,' 1848, vol. i, p. 306.—Boy aged 13. 1st day.—Depressed compound fracture on right side of vault, with escape of cerebral substance. Operation.

11th.—Convulsions and paresis of left side appeared, and to-day hernia cerebri is forming.

15th.—Hernia size of large orange. "The protrusion was cut through the centre, and the knife passed an inch down into the substance of the brain." No matter escaped.

16th.—Death.

Post-mortem.—Hernia composed of brain softened and contused, mixed with irregular coagula of blood. Beneath, brain was soft and pulpy and tinged with blood to the extent of $1\frac{1}{2}$ inches down and backwards from the surface. Meningitis. Secondary purulent deposits in lung.

Cases in which the Hernia was Removed; Recoveries.

CASE 69. J. G. de Merveilleux, jun., 'Lond. Med. Reposit.,' 1818, vol. ix, p. 283.—Boy aged 10 was wounded by a fork above the left frontal sinus. Two days later the wound was suppurating, and a probe passed four inches through the opening into the brain.

Phrenitis supervened at the end of a month, and three weeks later hernia cerebri formed and increased to the size of a pigeon's egg in spite of frequent applications of lunar caustic and other escharotics. Then it was ligatured, and in a fortnight sloughed off. Dry lint dressing. The wound was well in ten days, and remained so.

CASE 70. Webster, 'Lancet,' 1850, vol. i, p. 760.—A boy had to be trephined for a depressed fracture of the skull with laceration of dura mater and brain. Some protruded brain was removed. 'Hernia cerebri' soon supervened, and required excision. The case ultimately recovered, and was seen by Webster thirty years later. The patient had had good health since the accident.

CASE 71. Pring, 'Edin. Journ. ;' Stanley, 'Med.-Chir. Trans.,' vol. viii.—Case in which a protruded mass (regarded as real hernia cerebri) was removed, and pressure employed. Cure effected.

CASE 72. Stanley, 'Med.-Chir. Trans.,' vol. viii (1817).—Boy aged 11. 1st day.—Trephined for depressed fracture of frontal bone. Dura mater uninjured.

7th.—Cerebral hernia ; pressure flattened it out.

8th or 9th.—Superficial portion—cortex and medulla—removed. Pressure applied.

10th or 11th.—Protrusion increasing. Whole mass now pared off at level of skull (medullary substance). Pressure continued, but still there was a tendency to increase, which ceased in about four days. The putrid surface now sloughed, and granulation and healing took place in a short time.

CASE 73. Spring's paper, Obs. 55.—A child came under M. Ficker's observation with a cerebral hernia (size of a goose's egg) on the left parietal bone fourteen weeks after the accident that led to it. There was complete right-sided paralysis. First local applications were tried. On the second day after removal of the destroyed part pressure by strapping applied. On the ninth day it was diminished one third. Ligature was now attempted, but as it produced convulsive movements and other symptoms, it was taken off. On the fifteenth day a fresh ligature was applied, and gradually tightened, and on the thirty-first the tumour came away. There was some necrosis of the edges of the opening, but this was no obstacle to cicatrization, and healing was complete at the end of four months.

CASE 74. G. B. Mallet, 'Trans. Prov. Med. and Surg. Assoc.,' vol. vii, p. 343.—J. D—, aged 4. Compound depressed fracture of

upper and central part of the frontal bone, with injury to brain. Paralysis of left side. Broken and loose portions of bone removed. In a week hernia cerebri, which increased to size of a duck's egg in spite of pressure and local applications. Remained stationary for a fortnight, and was then removed by the knife. A spicule of bone was found projecting from the inner table, and was removed. After the operation there was a slight inclination to rise up, but it soon subsided, and the wound healed. The paralysis gradually disappeared. The tumour removed was evidently brain substance.

CASE 75. Longmore, 'Brit. Med. Journ.' 1882, vol. ii, p. 928.—September 10th, 1879.—Male aged 39. Gunshot wound of right temporal region.

28th.—Supervention of left hemiplegia. No loss of sensation.

October 7th.—Paralysis had disappeared.

February 11th, 1880.—Invalided home. Wound discharging slightly.

April 8th.—Bullet and loose bone, &c., removed.

July.—Small hernia cerebri with slender pedicle.

7th.—On probing by the side of the pedicle for loose bone "the tumour became quickly puffed up to the size of a marble, and giddiness, nausea, and headache were produced." These symptoms subsided on the following day, and the tumour returned to its previous size. Pressure could not be continued as it caused headache.

December.—Alarming cerebral symptoms. Status epilepticus. Operation. Ring of dura mater embracing pedicle was divided, and a piece of bone found firmly wedged between them. Brain explored for pus, none found. Hernia cerebri cut off. Incision in dura mater united. Rapid recovery.

January 2nd, 1881.—Wound healed.

CASE 76. G. B. Mallet, 'Trans. Prov. Med. and Surg. Assoc.' vol. vii, p. 343.—William H—, aged 12. Compound depressed fracture about the coronal suture (centre). Brain injured to depth of three-quarters of an inch. Paralysis of right side. Sight of right eye nearly gone. Loose portions of bone removed. In a week hernia cerebri began to form, and resisted all treatment till it measured 6 inches \times 3½ inches \times 2½ inches in elevation. Then removed by the knife as the patient was evidently sinking from constitutional irritation. Halfway through the knife encountered a small portion of loose bone. A few days later a disposition to protrude was controlled by pressure. The paralysis of leg began to

mend. The wound was healed in eleven weeks from the accident. Slight lameness remained. The eye had perfect vision, but the arm remained useless.

The tumour was composed of brain substance—not so firm as ordinary brain,—and had a cavity in its centre containing about an ounce of limpid serum. It was stated to be lined by a transparent membrane.

CASE 77. Allen, 'New England Journ. of Med. and Surg.' 1819, vol. viii, p. 323.—Man aged 19. A gun wad entered the brain about the posterior inferior angle of left parietal bone. Foreign substances were carefully removed. Hernia cerebri formed, and on the tenth day it closed the aperture in the bone, and prevented escape of discharges, and so led to pain, delirium and convulsions. Excised with relief of symptoms. Fungus cerebri gave no trouble, though caustic and moderate pressure were employed. Some months after the wound was "almost entirely sound."

CASE 78. Lawson, 'Lancet,' September 27th, 1890, vol. ii, p. 669.—May 13th, 1889.—Evacuation of subdural abscess due to necrosed bone six weeks after wound of left fronto-parietal region.

18th.—Hernia cerebri following symptoms.

August 6th.—Circumference of hernia cerebri 5 inches.

September 10th.—Pus found on probing hernia. Incision. Symptoms apparently improved.

October 11th.—Portion shaved off and pressure applied.

31st.—Hernia only slightly altered in shape.

November 3rd.—In consequence of further pressure symptoms, an intra-cerebral abscess seems to have been opened with a director, and hernia was burnt off on a level with the skull.

5th.—Hernia the size of a Tangerine.

December.—Caustics applied.

February 12th, 1890.—Sinus found to lead into abscess cavity 2½ inches from surface. Drained.

13th.—Brain symptoms.

15th.—Hernia rapidly increasing, but symptoms better.

21st.—Caustics as before. Hernia gradually decreased till it was quite flush with the skull.

May 9th.—Went out.

Three weeks later healed completely.

CASE 79. John Adams, 'Lond. Med. Gaz.,' vol. xxxiv, p. 336.—Reference to a case by Mr. Hill in which to prevent a repetition of

bad symptoms he was obliged to shave away the tumour, and to push a lancet into its root as often as stupor and other symptoms showed that matter was lodged there, by which the patient was uniformly relieved, and eventually recovered.

CASE 80. Mason, 'Lancet,' 1890, vol. i, p. 801.—September 19th, 1889.—Boy aged 6. Compound comminuted fracture of right frontal eminence from a horse kick. Portions of bone removed.

28th.—Hernia cerebri forming.

30th.—Rapidly increasing.

October 9th.—Size of Tangerine.

10th.—Sliced off level with skull.

22nd.—Larger than ever. Sliced off.

28th.—Again increasing. Firm bandaging caused slight but gradual diminution in size.

November 12th.—Level with surrounding skin. Wound healing.

January 20th, 1890.—Went out well.

CASE 81. Maclaren, 'Clin. Soc. Trans.,' 1886.—Man aged 26. 3rd day.—Depressed fragment of left frontal bone raised.

9th.—Loose fragments of skull removed, and hernia cerebri first cut off.

14th.—Right hemiplegia had developed.

23rd.—Hernia cerebri again cut off. Silver plate slipped inside skull.

42nd.—Hernia cerebri removed for the third time, and plate firmly fixed.

104th.—Plate finally removed.

A year later the leg had recovered, but the arm did not. Speech fairly good, but slightly affected.

CASE 82. Spring's paper, Obs. 45.—A youth of 14 developed a fungus after trephining for injury. A ligature caused it to fall off, but it was reproduced and was again similarly destroyed. Several times this was repeated until a mass the size of a fist had been removed. The patient, however, recovered.—Fabrice de Hilden.¹

CASE 83. Quesnay, 'Mem. of the French Academy of Surgery,' tome ii; Stanley, 'Med.-Chir. Trans.,' vol. viii. Young man. 1st day.—Fracture of right parietal bone and wound of brain.

2nd.—Convulsions. Paralysis of opposite side, fever, delirium;

¹ Fabrice de Hilden relates also several similar facts, of which several treated by caustics ended in death ('Observat. Chir.,' Centur iv, Obs. 3, p. 289).

injured brain black, swollen, and softened; protruded. Some of the projecting brain daily removed.

18th.—Fall from bed. The protruded gangrenous brain was detached and found in dressings. More gangrenous substance protruded through opening and was daily cut off.

35th.—When drunk patient tore away the protruded brain. Almost all the sloughy part was taken away. Recovery now set in, the exposed surface becoming red instead of black. The paralysis remained and he was subject to epileptic motions. Intellect perfect.

CASE 84. Van Swieten, 'Commentaria,' tome i, p. 440; Stanley, 'Med.-Chir. Trans.,' vol. viii, p. 37. Boy aged 14. Pus liberated by perforating skull two months after an injury. Hernia cerebri formed and cut off at its base by a thread. It was quickly reproduced and similarly removed. This was repeated several times until the mass removed equalled the size of a large orange.

The protrusion now ceased and the boy recovered.

CASE 85. J. W. Heustis, 'American Journ. Med. Sci.,' 1828 or 1829 (two papers). Negro aged 8. On 5th day after operation for fracture of left parietal bone in which splinters had entered the brain substance, showed tendency to develop a fungus. As soon as it protruded above the skin level it was shaved off, and this had to be repeated every two or three days. Then it was left alone for a short time, but rapid growth continued, and it was again sliced away and sponge pressure applied. It now ceased to grow, and soon gave place to a large excavation in the brain, which rapidly filled up, the patient recovering perfectly.

CASE 86. Porter, 'Dublin Med. Press,' 1839, vol. i, p. 54. Mr. Roney, in a case of hernia cerebri, day after day sliced off the fungus, which day after day was renewed. The man recovered, after losing more brain than his skull could at any time have contained.

Remarks by Mr. Porter at a society.

CASE 87. W. W. Stewart, 'Philadelphia Journ. of Med. and Phys. Sciences,' vol. xii, p. 205, 1826.—Boy aged 9. Trephined for extensive compound depressed fracture of left parietal bone with wound of brain, caused by a horse kick.

16th day.—Hernia cerebri began to form, coincident with a recurrence of paralysis in the opposite arm and leg, and an aphasic condition. The hernia reached the bulk of a middle-sized orange, and was practically left to nature.

35th.—Excision decided on. Removal in portions on four successive days. Protrusion continued, and the shaving-off plan was carried out at intervals for twenty days, when it showed signs of abating. A copious discharge oozed from the surface, and did not cease for three or four weeks. In the twelfth week the wound was healed, and the paralysis completely recovered in some months. Mental condition unimpaired.

The boy was well three years later.

Cases in which the Hernia was Removed ; Deaths.

CASE 88. Moyle, 'London Med. Reposit.,' 1817, vol. viii, p. 390. —Man aged 20. 1st day.—Compound depressed fracture of right parietal bone. Dura mater punctured by depressed bone. Trephined, and bone elevated.

4th.—Hernia cerebri grew to size of pigeon's egg.

11th.—Ligatured.

13th.—Tumour came away. No further protrusion.

24th.—Rigors, spasms.

33rd.—Pus escaped from brain, and when spasms came on it started out from a little hole in the direction of the fracture towards the ear.

40th.—Death.

No post-mortem, but superficial examination revealed nothing particular, except the abscess in the brain.

CASE 89. Cæsar H. Hawkins, 'Med.-Chir. Trans.,' vol. xxxix, p. 289.—Man aged 36, who had suffered from syphilis. On February 2nd, 1832, had a pulsating tumour 5 inches in diameter on the upper and posterior part of the right side of his head. From its centre a small fungus protruded (walnut) through an opening in the scalp. There were other depressions in the bone on the other side, through which brain pulsations could be felt. The disease had begun two and a half years before admission. A depression in the bone first formed, and subsequently the tumour took its place. Eighteen months before admission pulsation was first pointed out to the patient. After various punctures at different times the fungus protruded through one of them. The fungus was removed by ligature, and then the tumour was similarly dealt with. Serious symptoms supervened. He died on the forty-seventh day after admission, when the tumour was increasing.

Post-mortem.—There were large apertures in the posterior part of the skull, which was much thinned. The anterior part was thickened

and ivory-like. An opening from the centre of the fungus led into a large abscess in the right hemisphere, which had discharged itself during life. The disease had evidently begun in the bone.

CASE 90. Pearson, recorded by Stanley, 'Med.-Chir. Trans.,' vol. viii, p. 45.—November 12th, 1812.—Man aged 20, in Lock Hospital for syphilitic caries of frontal bone.

May 4th—12th, 1813.—Alarming head symptoms, relieved by treatment.

July 15th.—Necrosed bone easily removed. Hernia cerebri developed. No benefit from moderate pressure. Portions rendered loose by accident were cut away. The tumour increasing its base was ligatured, and tightened daily without very obvious effect.

October 25th.—A large portion in a corrupted state accidentally broken off,

27th.—Stupidity, insensibility; death.

Dimensions of tumour before the ligature $5\frac{1}{2} \times 6\frac{1}{2}$ inches, and 2 inches high.

Post-mortem.—An abscess containing between 2 and 3 ounces of pus was found in the anterior lobe of right hemisphere. Lateral ventricles contained some ounces of bloody fluid. The circumference of the hernia was much firmer than its centre. Opening in the bone $3 \times 2\frac{1}{2}$ inches.

CASE 91. John Duncan, 'Brain,' 1879–80, vol. ii, p. 413.—March 22nd, 1872.—Head injury.

May 11th.—Admitted with hernia cerebri ($3\frac{1}{2} \times 2 \times 1\frac{1}{2}$ inches) over left parietal bone. Aphasic; right side paralysed.

29th.—Serious symptoms set in; pyrexia, headache.

June 3rd.—Protrusion sliced off, and abscess cavity size of walnut exposed.

6th.—Death.

Post-mortem.—An abscess cavity occupied the superior parietal lobe; it contained diffuent brain matter and pus. The margins of an aperture in the bone were considerably depressed.

CASE 92. Adamson, 'Med. Press and Circ.,' 1874, December 30th, p. 565.—August 23rd.—Man aged 19. Compound fracture of left parietal bone with laceration of brain and membranes.

September 5th.—Semi-coma came on. Spicule of bone projecting into brain removed. Consciousness soon returned.

22nd.—Wound nearly closed, but seemed fuller.

26th.—Hernia formed. Right hemiplegia; vomiting.

28th.—Shaved off level with scalp.

October 8th.—Hernia entirely disappeared. Semi-coma.

11th.—Death.

Post-mortem.—Abscess in left anterior lobe which had almost destroyed the third convolution and injured others. Congestion on surface of right hemisphere.

CASE 93. Spring's paper, Obs. 54.—After the operation of trephining, when the brain had been injured, a hernia cerebri occurred. It was removed by a cutting instrument and the ligature, but grew again larger, and necessitated fresh operations at short intervals. Five or six ounces of brain matter were thus removed without accident. The tendency to reproduction did not diminish, and it was necessary to ablate nearly every evening. One night the patient was taken with delirium, shivering and insensibility, and died.

Post-mortem.—A large abscess had formed in the substance of the middle lobe, and had communicated with the ventricles, which were full of pus.

Reproduced from Chassaignac, 'Lésions traumatiques du Crâne,' Paris, 1842.

CASE 94. John Adams, 'Lond. Med. Gaz.,' vol. xxxiv, p. 337; Dr. Tuthill's case ('Lond. Med. and Surg. Journ.,' 1831).—Hernia cerebri followed trephining for compound depressed fracture of left parietal bone. Its appearance was preceded by a violent attack of inflammation. After excision by ligature it was reproduced. After a large quantity of cerebral substance had been lost, compression was employed, and immediately produced paralysis of the opposite side, which continued until pressure was remitted. Death three weeks after the convulsive attack.

Post-mortem.—Abscess of middle lobe communicating with the ventricle.

CASE 95. Stanley, 'Med.-Chir. Trans.,' vol. viii, 1817.—Boy aged 12. Trephined for depressed fracture of skull.

10th day.—Hernia cerebri protruded and grew to size of small orange in three days. Partial insensibility. It was sliced off, and death occurred in three days.

Post-mortem.—Brain soft and broken where cut off, the condition extending into its substance. There was slight protrusion through the opening. Lateral ventricles large, and filled with transparent fluid. Meningitis.

The tumour cut off contained cortex, medulla convolutions, sulci.

CASE 96. Stanley, 'Med.-Chir. Trans.,' vol. viii, 1817.—Boy aged 13. 1st day.—Depressed fracture of frontal bone from kick of a horse.

2nd.—Operation. Bone elevated, and 3 × 2 inches removed. Dura uninjured.

7th.—Bulging dura mater sloughed.

8th.—Hernia cerebri size of hen's egg.

9th and 10th.—Hernia cerebri increasing rapidly in spite of slight pressure.

10th—16th.—Removed at level of skull; firm pressure. Part removed composed of cortex and medulla, healthy in appearance. Surface sloughed, suppurated and granulated.

17th.—Drowsiness. Relaxation of pressure resulting in protrusion. Then restlessness and paralysis of left arm gradually developed.

24th—27th.—Considerable quantity of serous fluid (cerebro-spinal) constantly oozed from the centre of the protrusion, trickling down the cheek in a stream. Hernia cerebri exceeded a large hen's egg in size. Gradual decline, and death on twenty-seventh day.

Post-mortem.—Arachnitis. The brain substance between the hernia and the anterior cornu of the lateral ventricle was soft and pulpy, and conveyed the idea of rottenness. All around the protrusion the brain had separated from the dura for a considerable space, and the tumour itself was considerably lessened in all its dimensions.

CASE 97. Douglas, 'Brit. Med. Journ.,' 1874, p. 646.—Boy aged 18. August 5th.—Compound depressed fracture in region of anterior fontanelle with symptoms of compression. Removal of depressed fragments. Injury of right hemisphere by an embedded fragment.

6th.—Recovery from compression.

12th.—Suppuration. Piece of sloughing brain separating. Unconscious.

16th.—Conscious. Facial convulsive movements.

20th.—Hernia cerebri.

23rd.—General state improving. Hernia increasing.

28th.—Hernia increased in spite of pressure. Dimensions 3½ × 2½ inches, circumference 6½ inches. Catgut ligature applied.

September 3rd.—Weaker. Separation expedited by knife. Tumour composed of broken-down, sloughy material, pus and blood.

10th.—Much weaker. Hernia protruding, and again removed. No further protrusion. Patient gradually got worse.

27th.—Death.

Post-mortem.—A conical cavity extended vertically into the right hemisphere. Meningitis. Offensive odour.

CASE 98. Dr. J. Thomson, 'Cooper's Surgical Dict.,' 6th edit., p. 690.—A soldier (P). Wound of right frontal bone with spicules of bone driven in upon the brain. A large fungus protruded. In the progress of the case escharotics were applied, and portions torn off by the patient. The part exterior to the cranium was twice pared off by the knife.

Post-mortem.—The whole of the right hemisphere was found converted into a soft pulpy mass. The left hemisphere was normal.

CASE 99. Davidson, 'Edin. Med. and Surg. Journ.,' 1838, vol. 49, p. 41. August 1st.—Man aged 26. Trephined for compound depressed fracture of right parietal bone. Dura mater not injured.

29th.—Fungus is growing from the dura mater. Increase of left arm and leg paralysis.

September 3rd.—Removed with a scalpel. Consists of cerebral matter.

15th.—The cerebral fungus, which had recurred, was again sliced off. Removal of portions of exfoliated bone.

19th.—Further increase of tumour. Death.

Post-mortem.—The brain was disorganised throughout the whole of the middle and greater part of the anterior and posterior lobes. Corpus callosum involved, and the parts in the floor of the right lateral ventricles were converted into the same pulpaceous reddish substance as the rest of the hemisphere. Each lateral ventricle contained a quantity of limpid serous fluid. The immediate seat of the protrusion was of a greenish-purple hue.

CASE 100. Armour, 'Glasgow Med. Journ.,' 1831, vol. iv, p. 341.—A boy aged 15, after operation for a depressed compound fracture of the right frontal bone with injury to the brain and its membranes, developed a hernia cerebri on the 3rd day. On the 4th it was torn away level with the bone. On the 6th, having recurred, it was dug out considerably lower than the level of the cranium. Again recurring, it was shaved off for several days. In despair pressure was employed, but was given up because it caused compression symptoms. Then caustics were used till the twenty-third day, when the patient became insensible, had convulsions, and died.

Post-mortem.—The hernia had receded and left a deep cavity. The greater part of the substance of the right hemisphere was gone,

its remaining surface being in a putrid-looking state. The brain substance was soft and more pulpy than natural.

CASE 101. Porter, 'Dublin Med. Press,' 1839, vol. i, p. 114.—Six days after the accident, a man was trephined for fracture of the temple, pieces of bone being impacted in the brain. Next day a fungus formed and was removed. Reproduced to size of an orange; was accidentally detached. Five times the fungus reappeared, and was removed by the patient himself or came away in the dressings. Amount lost = 4 oz. His symptoms were—he appeared heavy, restless, answered questions unwillingly, had a slow pulse. He developed paresis of the opposite arm. After death the fungus, which had been prominent, began to sink until a hole $2\frac{1}{4}$ inches deep was left.

Post-mortem.—The loss of brain substance reached to the lateral ventricle. During life after removal of one fungus a deep hole was observed in the brain, from which a clear serous fluid escaped in large quantity over patient's neck and arm. A large portion of the anterior lobe was destroyed, and the substance of the brain all round was highly vascular, and so soft that it was easily broken down by a touch of the finger.

On the opposite hemisphere an abscess containing 1 ounce of pus existed between the dura mater and the brain.

CASE 102. Prescott G. Hewett, 'Lancet,' 1864, vol. i, p. 241.—Hernia cerebri developed after separation of a large sequestrum, due to syphilis, from the frontal bone. The dura mater gave way.

The protrusion covered the eye, and in three weeks reached as low as the angle of the mouth. It was cut away, and other portions of brain soon followed, and were similarly dealt with. So matters went on when paralysis of the right side made its appearance. The patient sank in about a week after this.

Another case died four days after removal of the temporal bone, which was followed by hernia cerebri.

CASE 103. Spring's paper, Obs. 46.—Bartholini in 1649 records the case of a child of 4, in whom extirpation of a double fungus was followed in a few days by death.

CASE 104. Gwynn, 'Dublin Med. Press,' 1841, vol. v, p. 181.—July 18th, 1837.—Man aged 25. Trephined for depressed compound fracture of the parietal bone, through which brain matter had been

forced, and which had produced paresis of the right leg and convulsions.

20th.—Hernia cerebri showed itself.

25th.—Compression symptoms with increase of hernia. Hæmorrhage took place from it, and to prevent death from bleeding it was cut off close to its overlapping base, and styptics applied on lint. The tumour quickly recurred, and evidently was septic. Slight recurrence of hæmorrhage.

28th.—Death.

The tumour cut off was the size of half an orange, and consisted chiefly of blood and brain, the latter external to the former, and seemed to form the parietes of a cyst into which blood had flowed and coagulated.

Post-mortem.—The parts of the brain in the vicinity of the fracture were soft, pulpy, and mixed with blood.

CASE 105. Moyle, 'Lond. Med. Reposit.,' vol. viii.—Boy aged 8. 1st day.—Compound depressed fracture involving both parietals. Operation. Large wound of membranes found. Escape of brain substance

6th.—Hernia cerebri; convulsions. Compression failed.

11th.—Tumour removed by the knife; the hæmorrhage nearly proved fatal.

13th.—Death.

CASE 106. Grinston, 'Lond. Med. Journ.,' 1789, vol. x, p. 277.—October 13th, 1788. Boy aged 4 was trephined for compound depressed fracture of right parietal and frontal bones, when the meninges were found injured, and portions of brain protruded. A few days later a fungus appeared, and was gradually reduced by solution of blue vitriol and pressure. Progress was satisfactory till early part of December, when it reappeared, and grew rapidly in spite of caustics and pressure, and was finally removed by ligature.

February, 1789.—Left facial paresis always relieved when matter escaped from the opening in the bones.

23rd.—Vomiting. Convulsions involving left arm and face. The fungus, which had grown as large as before, was incised, and an ounce of pus let out, but his condition continued unsatisfactory, and on March 11th further interference evacuated a large quantity of colourless fluid with some relief.

March 12th.—The prominence was removed, but no further fluid appeared. The tumour grew again rapidly, and would not bear pressure. Vomiting, spasmodic affections, especially a rigidity of

the neck, now made their appearance. The tumour gradually diminished, and became flaccid; yet the symptoms reduced him quickly, and he died on March 31st.

Examination of brain.—The wounds of the dura mater remained open. "The tumour had its origin from that membrane, to which it inseparably adhered. An empty cavity large enough to contain seven or eight ounces of fluid had been formed by the pressure of the tumour."

Remarks.—There is probably no doubt that this was a true hernia cerebri. Indeed, it is stated that the tumour resembled at one time brain, at another cartilage or steatoma. Death was probably due to meningitis, to which the operative procedures possibly contributed.

CASE 107. Van Swieten, Spring's paper, Obs. 49.—A fungus appeared on the fifth day in a boy of 7 after a fracture of the right parietal. It was treated by exsiccant remedies, and persisted for three months. At the beginning of the fourth month there was a considerable increase. It was then destroyed by caustics. In twenty-four hours it recurred bigger than before, and there was a return of cerebral symptoms. Caustics proving useless, the tumour was got rid of by ligature. This was followed by serious cerebral symptoms, and by the growth of a third tumour as big as a nut. In twelve days this had subsided and left an excavation in the brain substance. Two days later this was filled by a fourth fungus, and in a few more there was opisthotonos and death.

CASE 108. Stephen Smith, 'New York Journ. of Med.,' 1857, 3rd ser., vol. ii, p. 83.—Man aged 40. 1st day.—Trephined for compound depressed fracture of right parietal bone. Dura mater found injured, and brain substance escaped.

4th.—Paralysis of left arm. Convulsions on paralysed side.

5th.—Fungus cerebri.

10th.—Fungus large. Convulsions have been frequent.

14th.—Paralysis has disappeared.

15th.—Fungus removed. No brain substance found in it. Composed of fibrin and hæmatoidin.

19th.—Removed again.

21st.—Removed again (possibly after sloughing).

22nd.—Paralysis of left arm came on.

33rd.—Condition unfavorable. Fungus reappeared, and continued to grow.

35th.—Paralysis extended to whole of left side.

37th.—Death with all the symptoms of abscess of brain.

No autopsy.

CASE 109. Weir, 'New York Journ. of Med.,' 3rd ser., vol. vii, p. 291, Case 5 (practice of Dr. Buck).—Girl aged 6½.

May 22nd.—Depressed fracture of the left side of the frontal bone.
26th.—Trephined.

June 8th.—Hernia cerebri appeared, forcing open wound and the dura giving way.

17th.—Size of a hen's egg. Excised by ligature and scissors. (Cerebral substance. Microscope: no nerve tissue found, but cells and granules in quantity.)

23rd.—A jet of clear fluid forced from opening in the centre of the exposed brain, during crying. This was repeated several times, and it was supposed to come from the lateral ventricle.

28th.—Death. No autopsy, but the brain receded, leaving a hollow that would receive an egg, and the lateral ventricle opened into its floor.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 108.)

NON-TUBERCULOUS POSTERIOR BASIC MENINGITIS IN INFANTS

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So large a proportion of all the cases of meningitis in children are tubercular that we are apt almost to ignore the existence of other forms; nor is this so surprising when we find that, whereas the tubercular disease is one running a tolerably definite course, and having a very well defined pathological anatomy, the other and rarer forms of meningitis present the greatest diversity, alike in course, symptoms, and morbid appearances. Some cases are very acute, lasting only a few days; others extremely chronic. In some the vertex is mainly affected, in some the base, whilst in others the affection is uniformly distributed over the whole of the brain and perhaps the spinal cord also. Even more diverse is the reputed causation of these varied conditions—injury, ear disease, rheumatism, exposure to the sun, congenital syphilis, the acute specific fevers, influenza, pneumonia, septic conditions, &c., are all assigned as possible causes, and even then some cases remain which we have to be content to term idiopathic. Amid this chaos I wish to single out a

certain number of cases which present a fairly definite course and symptomatology, and tolerably uniform pathological changes, and which are therefore probably due to some one definite cause, though this as yet eludes our observation. The cases referred to are those of non-tubercular meningitis, more or less chronic in character, and affecting a definite region at the base of the brain in young children.

So far as I know, this disease has only been at all fully described previously in a paper contributed by Dr. Gee and Dr. Barlow to the 'St. Bartholomew's Hospital Reports' for 1878 (vol. xiv), entitled "The Cervical Opisthotonos of Infants;" but, as the title of the paper indicates, they regarded the condition from the standpoint rather of the chief symptom, the opisthotonos, and described twenty-five cases in which this was present, although in all those in which an autopsy was obtained basic meningitis was found. Several of their cases were seen only once or twice as out-patients, and the ultimate result was not ascertained. On the other hand, I wish, if possible, to describe this basic meningitis as a definite pathological condition, with more or less characteristic symptoms and course, basing the description on a few cases under observation in hospital, and followed out to an autopsy.

The cases to be recorded—except perhaps No. 11—may, I think, be regarded as fairly typical ones; others showing less characteristic symptoms or less definite morbid changes have been met with, but, in describing a comparatively rare and not very commonly recognised condition, it seems well to limit our attention first to the typical cases, and once having noted the usual manifestations we are in a better position for dealing with less definite examples of the disease. Such irregular, atypical, and border-line cases are sure to be met with,—in what disease do they not occur? Little good, however, is, I think, to be gained from dwelling upon them at first, although they may prove interesting later, for we have to begin by establishing, if possible, the identity of the disease, then to ascertain its relations with other

morbid conditions, and afterwards to learn how to recognise it in its less characteristic manifestations.

All the cases occurred in the Victoria Hospital for Children, Chelsea. I am indebted to my colleagues Dr. Ridge-Jones and Dr. Drewitt for the use of the notes of the cases during life, and to Mr. Holmes Spicer, ophthalmic surgeon to the Hospital, for most of the reports upon the ophthalmoscopic appearances.

Both the clinical and pathological records of the later cases are more complete than those of the earlier ones; this is owing to the fact that it was not till I had met with several examples of the disease that I became specially interested in it, and so led more fully to investigate the symptoms and morbid changes.

[In each case the age given is that of the child when admitted to the hospital.]

CASE 1.—Sidney K—, aged 8 months, admitted March 5th, 1890. Six other children living and well; one miscarriage. No previous illness, but said to have had snuffles. Fed on breast only. Stated to have been ill since a fall on the top of the head on December 20th, 1889; had convulsions and vomiting next day, and occasional convulsive attacks after, but had had none for a fortnight until the day before admission, when he had a severe attack followed by sickness. On admission, head greatly retracted; fontanelle rather bulged; slight rigidity of limbs and twitchings of face; occasional squint; pupils equal; pulse about 100, rather irregular; severe vomiting; bowels open. Temperature on admission 96° , rose to 102° , but fell again before death, which, without any change in symptoms, occurred on March 8th.

Autopsy (five hours after death).—Body extremely emaciated; slight beading of ribs, and very slight craniotabes in cerebellar fossæ. Brain: convolutions much flattened, dry, and bloodless. At the base all the structures from the posterior border of the cerebellum to the optic commissure firmly matted together by a very thick layer of

lymph; also much matting together in both Sylvian fissures. Lateral and third ventricles and aqueduct of Sylvius enormously distended with fluid, but showed no sign of inflammation. Brain substance almost diffuent. Left middle ear healthy, right full of inodorous pus; membrana tympani intact. Cranial bones and dura mater healthy. No thrombosis in sinuses. Other organs normal; no sign of tubercle anywhere.

CASE 2.—Alfred S—, aged 5 months, admitted May 10th, 1890. Fourth child. No evidence of syphilis in family history or in child's condition. Fed on breast only. Quite well till April 20th, when taken ill suddenly; said to have become unconscious and motionless, then constantly sick for two or three days, and had sudden screaming fits. For about a fortnight before admission had head retraction and convulsions every day. On admission, head retracted; anterior fontanelle bulging; pupils equal; slight squint; pulse 108, regular; sighing breathing; food taken well, no vomiting. Whilst in the hospital there was excessive head retraction and some general opisthotonos; anterior fontanelle tense; little or no rigidity of limbs, but twitchings and slight convulsions occasionally; squint and nystagmus at times; pulse rapid (140 to 160) and regular; breathing sometimes irregular; sickness most days; bowels open regularly; no optic neuritis. Temperature normal, save for an occasional evening rise to 100° , but during the last two days of life it rose steadily and reached 105.8° at death, which occurred on May 25th. The head retraction and the rigidity of the neck lasted about one hour after death. Total duration of illness thirty-five days.

Autopsy (twenty-five hours after death).—Body considerably emaciated; very slight cranio-tabes in cerebellar fossæ, but no other sign of rickets or of syphilis. Brain: convolutions flattened, and cortical veins rather congested; pia mater at base thickened, with matting together of parts over the central portion of the inferior surface of

the cerebellum, and over the medulla, pons, and base of the cerebrum, as far forwards as the optic commissure; also over the tip of the left temporo-sphenoidal lobe, where there was a thin layer of puriform lymph. The fold of pia mater closing the fourth ventricle between the medulla and cerebellum thickened to form a firm membrane. Lateral ventricles dilated to form two large bags filled with turbid fluid containing flakes of lymph; fourth ventricle also much dilated. Brain substance almost diffuent. Middle ears and cranial bones healthy. Other organs normal, except for a cyst in the right kidney; no sign of tubercle anywhere.

CASE 3.—Emma H—, aged 7 months, admitted November 17th, 1890. First child, no miscarriages. No previous illness; said to have had snuffles soon after birth, but no rash. Fed on breast only. Five weeks before admission had diarrhoea for a week, and a week later became dull and restless when asleep; no vomiting. Convulsions, preceded by screaming for a day or two, set in twenty-five days before admission, right side most affected; increasing retraction of head noticed after first fit. On admission, head markedly retracted; frequent slight twitchings of face; no rigidity of limbs, but hands closed; right internal squint; pupils equal; no optic neuritis. In hospital there was extreme retraction of head throughout; no note of fontanelle; no convulsions, and only very slight rigidity of limbs, except occasionally of the forearms; chewing movements, yawning, and sighing noted; pulse rapid (130 to 170), only once or twice noted to be slightly irregular; Cheyne-Stokes breathing; frequent vomiting; bowels relaxed. Temperature normal, except for one rise to 101.4° , but rose steadily before death on December 1st to 106.4° . Duration of symptoms five to six weeks.

Autopsy (thirty hours after death).—Body fairly nourished, no rickets. Brain: convolutions much flattened, dry, and bloodless. A thick layer of lymph extended over the base of the brain as far forwards as the optic

commissure, with matting together of all the parts there and along the Sylvian fissures ; lymph thickest over the pons. Thickening and opacity of the pia mater over the central part of the under surface of the cerebellum, ending on each side in a sharply-defined line. Isolated patches of lymph on the tip of each temporo-sphenoidal lobe, and a small patch also on the tip of each frontal lobe. The changes on the two sides very symmetrical. A few small isolated patches of lymph on the vertex, chiefly along the branches of the middle cerebral arteries. Pia mater closing fourth ventricle thickened, and foramen of Majendie apparently obliterated. Lateral and third ventricles and foramen of Munro much dilated, contained a few flakes of lymph (one large one on choroid plexus), and about 7 ounces of slightly turbid fluid, sp. gr. 1013, which did not coagulate spontaneously, but gave a large deposit of albumen on boiling, and contained abundant chlorides but no copper-reducing substance. Brain substance very soft. Right middle ear healthy, left contained a little non-fœtid, semi-purulent mucus. No disease of cranial bones ; no thrombosis of sinuses. Upper half of spinal cord almost diffuent, and protruded when dura mater was cut through ; lower half appeared healthy. Other organs normal ; no sign of tubercle anywhere.

CASE 4.—Alice O—, aged 3 months, admitted September 27th, 1892. Second child ; elder one healthy ; no previous illness ; no evidence of syphilis. Fed on breast only. Brought to the out-patient department on September 6th for sickness and screaming.

September 13th.—Marked head retraction.

20th.—Head retraction very marked ; constant sharp cry.

27th.—Symptoms as before. Child hardly able to take breast ; cries when head is moved.

On admission, fairly nourished ; extreme retraction of head ; fontanelle normal ; pupils equal ; frequent sharp cry ; fed with difficulty. In hospital marked bulging of the

fontanelle developed, and coincidently with this the head retraction diminished; no convulsions; some strabismus; no note of pulse; frequent vomiting; considerable diarrhoea, perhaps due to treatment by calomel. The child had to be fed by nasal tube. The optic discs were quite normal two days before death. Temperature normal except for an occasional rise to 100° or 101° ; no ante-mortem rise. Death on October 12th. Duration of illness about six weeks.

Autopsy (twenty-five hours after death).—Body much wasted; no rickets. Examination of head only permitted. Brain: convolutions pale and much flattened. A thick layer of purulent lymph covered the medulla, pons, and under surface of the cerebellum; considerable thickening round the optic commissure, but very little lymph and no distinct adhesions in the Sylvian fissures; a very thin layer of lymph over the tip of each temporo-sphenoidal lobe, and on the under surface of the frontal lobes. The fold of pia mater closing the fourth ventricle was greatly thickened, but presented in the middle line a circular aperture a quarter of an inch in diameter, with a well-defined edge. The lymph did not extend down the cord. Lateral and third ventricles and aqueduct of Sylvius greatly dilated; contained about 6 ounces of clear fluid, sp. gr. 1010. Choroid plexuses normal; many dilated veins on walls of lateral ventricles. Brain substance rather soft. Middle ears and cranial bones healthy. No tubercles seen.

CASE 5.—William N—, aged 9 months, admitted May 16th, 1892. Five other children; two living, three said to have died in infancy from convulsions. Child fed on breast only, and said to have been quite well till April 10th, when it was taken to a medical man for "a cold on the chest," and he noticed bulging of the fontanelle. Three days later patient had a convulsion, and two more shortly after; vomiting, head retraction, and rapid wasting supervened. On admission there was marked head retraction; circumference of head $18\frac{1}{2}$ inches; no optic neuritis. In

hospital the head retraction continued to be extreme, and the fontanelle became increasingly tense; no convulsions; nystagmus and squint were present. Pulse 100 to 120, regular; vomiting frequent; bowels freely open; temperature normal. On account of the extreme bulging of the fontanelle the lateral ventricles were tapped six times between June 6th and August 27th by a Southey's tube introduced at the side of the fontanelle, from 2 to 5 ounces of fluid being withdrawn each time, 20 ounces in all. The fluid was perfectly clear and watery in appearance, sp. gr. 1004 to 1006, contained a very faint trace of albumen, abundant chlorides, no copper-reducing substance. After the first tapping, when 4 ounces were withdrawn, the temperature rose to $100\cdot6^{\circ}$, but on no subsequent occasion was there any rise, and no ill effects resulted, even the pulse being unaffected. Attempts to apply pressure had to be abandoned owing to rapid ulceration of the scalp. Despite the tapplings the head steadily got larger, the circumference increased to 20 inches, and the interparietal suture opened throughout its entire length; nevertheless the child's condition began slowly to improve, in three months (June 29th to October 2nd) the weight increased from 12 lbs. to 15 lbs. 10 oz., the vomiting ceased, and by the beginning of August there was hardly any head retraction and no nystagmus; the child seemed more lively and took food better; bowels still rather loose; temperature normal. Iodide and bromide of potassium, a grain of each, were given three times a day.

All symptoms of active meningitis having subsided, whilst the hydrocephalus continued to increase, and would evidently in time cause permanent atrophy of the brain, the question of operative interference was considered. On the assumption that the hydrocephalus might be due to a blockage of the cerebro-spinal aperture by thickening of the pia mater between the medulla and cerebellum, a sequel to basic meningitis, it was thought that if a small opening could be re-established in this membrane the condition might possibly be relieved, especially as all active

inflammatory mischief seemed to have passed off. Accordingly on October 7th Mr. Raymond Johnson made an incision over the occipital bone, and trephined as far back as possible in the middle line, afterwards enlarging the aperture with forceps up to one third of an inch from the foramen magnum. The dura mater bulged into the wound and was opened just to the left of the middle line, several ounces of clear fluid escaped, and the cerebellum was clearly seen with its lobes rather widely separated by a deep space ; no fold of pia mater was visible. A small rubber drainage-tube was inserted, the wound sewn up, and an antiseptic dressing applied. Fluid continued to escape in large quantities, vomiting and then convulsions set in, the temperature rose to 104° , and the child died, twenty-eight hours after the operation, with a temperature of 102° .

Autopsy (forty-six hours after death).—Body wasted ; slight rickets. Brain : a good many adhesions between the dura mater and the brain, beneath the anterior fontanelle, the result doubtless of theappings. Examination of the brain was difficult owing to its almost diffuent condition, but evidences of old meningitis were found at the base in the form of numerous distinct white patches in the pia mater over the tip of each temporo-sphenoidal lobe, especially the left, and slight thickening around the optic commissure ; no distinct thickening could be made out more posteriorly, certainly not between the cerebellum and medulla. Convolutions injected and somewhat flattened. Lateral and third ventricles and aqueduct of Sylvius greatly dilated, with marked engorgement of vessels on the walls of the former ; no thickening of the ependyma ; choroid plexuses normal ; fourth ventricle apparently very little dilated. No tumour. Middle ears and cranial bones healthy. Other organs normal ; no sign of tubercle anywhere.

CASE 6.—Rose L—, aged 6 months, admitted March 20th, 1893. Fed on breast only. Illness began March 7th or 8th with convulsive attacks lasting some hours ; these

recurred on succeeding days, and the child vomited each time after being fed. The child was seen as an out-patient on March 13th; the pupils were unequal, there was no optic neuritis. On admission, a week later, the child was well nourished; head much retracted, with considerable general opisthotonos; anterior fontanelle bulging; pupils unequal; discs blurred; knee-jerks exaggerated; no rigidity; pulse 160; respirations 28. The lateral ventricles were tapped through the anterior fontanelle four times between March 23rd and May 2nd, from 2 ounces to 5 ounces being withdrawn each time, about 14 ounces in all. No ill effects followed; on the contrary, the pulse seemed to improve, and after the first tapping the respiration, which had been Cheyne-Stokes in character, became more regular; fluid clear, sp. gr. 1005 or 1006, contained only a trace of albumen. Despite the treatment the head slowly increased in size, and the child became more unconscious, having to be fed by a nasal tube from shortly after admission. Head retraction was excessive to the last. There were short convulsive attacks on March 30th, April 1st and 2nd; no squint; pupils always unequal; pulse rapid (120 to 160) and regular; respiration rather irregular; vomiting frequent, especially at first; bowels rather loose; some rigidity of the limbs was noted a few days before death. Temperature normal, only twice rose to 100°; no rise before death, which occurred rather suddenly on May 5th. Duration of symptoms about eight weeks. Autopsy refused.

CASE 7.—Arthur A—, aged 3 months, admitted May 9th, 1893. Second child, the other healthy; no evidence of syphilis obtained. Brought up on breast, and had vomited frequently from birth; had otherwise been in good health until ten days before admission, when he had a fit, followed by diarrhoea, head retraction, and some rigidity of the limbs. On admission, child fairly nourished; head markedly retracted; all limbs flexed and rather rigid; slight squint; pupils equal and reacted to light; knee-jerks and superficial reflexes normal; temperature 100·6°.

In hospital there was extreme head retraction throughout until death; fontanelle full, but not markedly bulged; no convulsions; pupils equal and contracted; pulse rapid (120 to 160), only slight irregularity, dependent probably on respiration; marked Cheyne-Stokes breathing; frequent vomiting; severe diarrhoea; child had to be fed entirely by nasal tube; cried much when disturbed. On June 2nd hands clenched, but limbs not rigid; knee-jerks brisk; tendency to ankle-clonus on left side; plantar reflexes present on both sides. Shortly before death, Mr. Spicer found distinct neuritis of the right disc; the left could not be seen. Temperature rose to 101.6° the day after admission, was afterwards normal, but reached 102.6° before death on June 5th. Duration of symptoms about five weeks.

Autopsy (twenty hours after death).—Body much emaciated; very slight rickets. Brain: convolutions notably pale and flattened. A firm membrane of thickened pia mater completely closed the fourth ventricle between the medulla and cerebellum; the thickening extended along the under surface of the cerebellum for not more than half an inch on each side of the middle line, ending in a tolerably well-defined margin; slight thickening also over the pons, and as far forwards as the optic commissure. There was a thin patch of lymph over an area fully half an inch in diameter on the tip of the right temporo-sphenoidal lobe, and traces of lymph on the tip of the left, but very little elsewhere. No thickening or adhesions in the Sylvian fissures or round any of the cerebral arteries. Lateral and third ventricles much dilated, and contained over 6 ounces of clear fluid, sp. gr. 1012; moderate dilatation of veins on walls of lateral ventricles, and a few small flakes of lymph. Choroid plexuses normal; brain substance soft. Middle ears and cranial bones healthy. No thrombosis of sinuses. Spinal cord rather soft, but no pus or lymph seen in spinal canal. Other organs normal; no sign of tubercle anywhere.

CASE 8.—Herbert B—, aged 6 months, admitted March 6th, 1894. Twelfth child, seven others living ; no miscarriages. No history of syphilis, ear disease, or injury obtained. Fed on breast only. Was taken ill on January 11th, and admitted to hospital January 16th, suffering from bronchitis and broncho-pneumonia. The child was noticed to have head retraction and a bulging fontanelle, with twitching of the fingers, squint, and Cheyne-Stokes breathing at times. Most of these symptoms, however, disappeared when the temperature fell to normal on January 22nd, but the head retraction persisted, and there was occasional vomiting ; no optic neuritis. The child was discharged on February 7th, the lungs being clear, but he continued to have head retraction and bulging of the fontanelle, and to be very fretful, and severe vomiting having set in he was readmitted on March 6th, much wasted, drowsy, the head retraction well marked, and the fontanelle much bulged. Head measured 17 inches in circumference. In hospital child stuporous, crying only when disturbed ; excessive head retraction with general opisthotonos ; fontanelle bulged ; no convulsions, squint, or nystagmus ; pulse rapid (110) and regular ; breathing regular ; some vomiting at first ; bowels regular ; no optic neuritis ; knee-jerks not obtained. Temperature 100.2° on admission, afterwards normal.

On March 12th, the circumference of the head having increased to 18 inches, Mr. Johnson trephined half an inch to the right of the middle line of the occipital bone, as close to the foramen magnum as possible. The dura mater bulged and did not pulsate ; it was incised and the cerebellum protruded ; on introducing a director towards the middle line, clear fluid escaped freely. The dura mater was left open and the bone not replaced, a horsehair drain inserted, and the external wound stitched up. The fluid was examined by Dr. Frank Blaxall, and streptococci in pure culture were found in it and also in the blood.¹

¹ Dr. Blaxall tells me that the streptococcus he found was similar in morphological and biological characters to the ordinary *Streptococcus*

The pulse was 120 to 130 both before and after the operation, breathing quite regular, but became very shallow. At the end of the operation the fontanelle was depressed and the bones overlapped; firm pressure was applied over the dressing. Twitchings came on very soon, but no general convulsions; the fluid soaked through in a few hours, and some packing was applied. The temperature rose in a few hours to 104.6° , and remained high, once reaching 105.8° , but was readily, though only temporarily, lowered by sponging.

On March 14th the child took more notice, its cry was more natural, and there was hardly any head retraction, but severe sickness set in, with Cheyne-Stokes breathing and more head retraction. On March 16th the wound was dressed and seemed to have ceased draining; fontanelle level but not bulging; pulse 150, regular. After this the temperature still continued high, the wound broke down, there was a little head retraction, the child gradually got weaker, and died on March 20th.

Autopsy (two hours after death).—Body moderately wasted; no rickets. Fontanelle admitted four fingers; interparietal suture loose, but not open; no bossing; no craniotabes. Trephine hole about an inch from margin of foramen magnum; wound completely broken down. Dura mater healthy. Brain: two changes observable over the surface, a chronic and very marked thickening of the pia mater, chiefly at the base, and a more diffuse recent purulent inflammation. Parts at base, from lower end of medulla to optic commissure, extremely thickened and matted together; on the under surface of the cerebellum the thickening ended in a fairly defined margin about three-quarters of an inch from the middle line. Cerebellum and pons firmly glued together, but in the middle line there was an opening in the thickened membrane about the size of a pin's head, through which fluid passed freely. A good

pyogenes and the *Streptococcus erysipelatis* (Fehleisen), except that this particular streptococcus curdled milk, which the others do not, and continued to do this for nine successive generations.

deal of lymph on the tip of the right temporo-sphenoidal lobe, and a little on the left. In the Sylvian fissures only slight adhesions due to recent puriform lymph, which extended all over the thickened part of the pia mater, and a little way on to the vertex. Lateral ventricles much dilated, and contained semi-purulent fluid; walls intensely injected and thinly covered with lymph; third and fourth ventricles also greatly dilated. Choroid plexus much swollen; veins of Galen apparently recently thrombosed. Brain substance rather soft, and posterior part of right lobe of cerebellum (beneath trephine wound) quite diffuent. Middle ears and cranial bones healthy. Some recent clot in right lateral sinus, but otherwise all the sinuses normal. Spinal cord appeared perfectly firm and healthy; central canal not dilated; no thickening of membranes. All other organs healthy; no sign of tubercle anywhere. Dr. Blaxall examined some of the thickened pia mater from the base of the brain for tubercles, but none were found.

CASE 9.—Julia C—, aged 12 months, admitted December 24th, 1894. Parents healthy; three other children alive and well. Soon after birth child said to have had snuffles and a rash, which soon got well. Never any otorrhoea. Had been mainly breast fed till ten months old, and was healthy until two months before admission, when she had measles; afterwards, for about six weeks before admission, she had head retraction, was very fretful, took food badly and wasted; no vomiting; became drowsy day before admission. There was an indefinite history of a fall on the head about two weeks before illness began.

On admission child pale, wasted, and drowsy; head moderately retracted; pupils much contracted. In hospital had marked head retraction throughout; stuporous, but could be roused, and would take food to the last; no convulsions, twitchings, rigidity, squint, or nystagmus; no slowing or irregularity of pulse, and no Cheyne-Stokes breathing noted; had attacks of vomiting and diarrhoea at times; no optic neuritis two weeks before death. Tempe-

perature rose at times to 101° (? due to bronchitis), and at death on January 31st to 104° . Probable duration of symptoms about eleven weeks.

Autopsy (twenty-one hours after death).—Body excessively emaciated; no rickets; six teeth; fontanelle nearly closed; no bossing or cranio-tabes. Brain: convolutions pale and slightly flattened. A good deal of yellowish lymph about the pons, and a little on the fold of pia mater closing the fourth ventricle, but no general thickening of this membrane, and certainly no closure of the foramen of Majendie. Very slight thickening of pia mater on under surface of cerebellum on each side of middle line. Large patch of lymph on tip of left temporo-sphenoidal lobe, and a much smaller one on right; also a few flakes about the top of the right fissure of Rolando. Sylvian fissures scarcely more adherent than normal. Lateral, third, and fourth ventricles considerably dilated, and contained about three ounces of clear fluid, which showed only a trace of albumen, probably due to a little blood accidentally mixed with it. Many dilated veins on walls of lateral ventricles. Choroid plexuses and velum interpositum apparently normal; veins of Galen not thrombosed. Brain substance of about normal consistence. Middle ears contained only a little thick mucus. Cranial bones healthy; no thrombosis of sinuses. Spinal cord and membranes apparently healthy; no lymph on surface; no softening; central canal not dilated. Other organs normal; no sign of tubercle anywhere. The thickened pia mater at the base of the brain was examined microscopically for tubercles, but none were found.

CASE 10.—Margaret F—, aged 11 months, admitted April 29th, 1895. Very little history was obtained; two other children were living and well, and there had been one miscarriage before patient was born. No history of rash, snuffles, or injury. The child had been well until three months before admission, since then had been sick at times and had gradually wasted. There are no notes of the child's

condition on admission, but on May 10th there was extreme head retraction, and the fontanelle was rather full; limbs excessively rigid, the legs and ankles being extended, the arms extended and pronated, the wrists and fingers flexed; no clonic spasm; slight external squint; no nystagmus; distinct optic neuritis; pulse rapid and regular; sick every day; child unconscious, but moaned when touched; swallowed fairly well.

May 24th.—Extreme head retraction and rigidity as before; fontanelle very full; no convulsions; breathing regular; food still taken fairly well. The coma gradually deepened, and the child had to be fed by nasal tube; the fontanelle became more bulged, the vomiting ceased. The other conditions remained unaltered, but the child became excessively emaciated; bowels freely open throughout. Temperature 97° to 100° , with an occasional rise higher, once to 103° . Death occurred on June 25th.

Autopsy (fifteen hours after death).—Body excessively emaciated; no rickets; no bossing; no craniotabes; four teeth. Head not obviously enlarged, but fontanelle admitted four fingers. Brain: convolutions pale and much flattened. Much thick lymph at base, reaching from lower end of medulla to front of optic commissure; very little lymph or matting together in Sylvian fissures. Space between medulla and cerebellum completely closed by a dense firm membrane, which extended along the under surface of the cerebellum for about half an inch on each side of the middle line, tapering posteriorly. Hardly any lymph on upper surface of cerebellum, but small patches scattered over cerebrum, chiefly in the Rolandic areas; small patches also about the tips of the temporo-sphenoidal lobes, but no distinct accumulation there. Veins of Galen not thrombosed, but considerable thickening around them in the great transverse fissure. Lateral and third ventricles much distended by about half a pint of clear fluid; some dilated veins on their walls; no lymph on choroid plexuses. Brain substance soft and oedematous. Middle ears normal, except for a little thin muco-pus in

left. Cranial bones and sinuses normal. Spinal cord: membranes normal; slight vascularity of surface of cord; substance fairly firm in upper half, but below mid-dorsal region almost diffuent. Other organs normal, except for some bronchitis and pulmonary collapse; no sign of tubercle anywhere. No tubercles were found on microscopic examination of the thickened membrane from the base of the brain.

CASE 11.—Florence M—, aged 7 months, admitted May 31st, 1895. Three other children alive and well. No evidence of syphilis; never any otorrhœa. Fed on breast only. Ailing for two months before admission, and had been treated as an out-patient for seven weeks at the West London Hospital; cried out a good deal, frequently sick, very drowsy, squint, rigidity at times. In hospital had very few symptoms; no head retraction or convulsions; took very little notice, but was not unconscious till three-quarters of an hour before death. Had squint and ptosis at first, but these subsequently passed off. Vomited occasionally, and for two days before death was constantly sick and very restless. Bowels freely open every day. Temperature 97° to 100° , with an occasional rise to 103° , and once to 105° (June 16th). Death occurred rather unexpectedly on June 27th.

Autopsy (twenty-three hours after death).—Body fairly nourished; slight beading of ribs; no teeth. Distinct craniotabes in posterior part of parietal bones, and in occipital fossæ; fontanelle widely open, admitting four fingers, and cranial sutures slightly separated. Brain: convolutions pale and markedly flattened. At base very slight thickening of pia mater and matting together of parts; a few small flakes of lymph here and there, notably on tip of left temporo-sphenoidal and of right frontal lobes. Medulla drawn up to and distinctly adherent to cerebellum, but foramen of Majendie probably not obliterated. Lateral, third, and fourth ventricles greatly dilated, and contained about half a pint of clear fluid,

having sp. gr. 1009, and containing a small quantity of albumen, probably due to accidentally admixed blood. Veins on walls of lateral ventricles considerably congested. The choroid plexuses and velum interpositum appeared normal, and the veins of Galen were not thrombosed, but there was some thickening and a little lymph about the pia mater in the great transverse fissure. Brain substance soft and oedematous. Middle ears healthy. Cranial bones and sinuses normal. Spinal cord and membranes firm and healthy. Other organs normal; no sign of tubercle anywhere. On microscopic examination no tubercles were found in the thickened membrane from the base of the brain.

Course and symptoms.—Although the cases described may be too few to make any very general deductions from, yet in reviewing them one cannot but be struck by a certain general likeness in them all in the symptoms and course of the disease, and by the still greater similarity in post-mortem appearances; whilst the great resemblance alike in clinical and pathological features between my cases and those recorded by Dr. Barlow indicates that we are both describing the same condition.

All my cases occurred in infants of from three to twelve months old (Dr. Barlow had one case aged nineteen months), in whom, as a rule, there was no evidence of malnutrition or of previous illness.

The onset varied, in some being sudden, in others gradual, but in nearly all head retraction and vomiting were the most prominent early symptoms. A definitely sudden commencement seemed to be indicated by one or more convulsive attacks, but on careful questioning it appeared that in several of these cases there had been indefinite symptoms of illness for a few days before, and it is perhaps most probable that such slight symptoms are usually if not always present, but in a baby may readily be overlooked until a convulsive attack first attracts attention to the child's condition.

Head retraction was not merely an early symptom, but

was also the most marked and persistent throughout; in only one case (11) was it absent. It was often of the most extreme character (far more than is usual in tubercular meningitis), the head being arched back to a right angle with the spine, and was sometimes accompanied by marked general opisthotonos. In two cases (4 and 5) the retraction diminished coincidently with increasing bulging of the fontanelle, and the same thing happened also in one of Dr. Barlow's cases, but there does not appear to me to be any causal relationship between the two, as in all my cases there was a large effusion of fluid in the ventricles with more or less bulging of the fontanelle, and yet head retraction usually persisted until death. Its cessation seems rather to indicate that the more active inflammation at the base of the brain is coming to an end; this certainly was so in one of my cases (5), and Dr. Barlow's also was one in which symptoms had persisted for some months, and in which post mortem the changes were evidently of old standing.

Slight twitchings in different parts of the body were frequently noticed, but general convulsions, except at the onset, seemed to be uncommon, as would in fact be expected from the limitation of the disease to the base of the brain; and probably for the same reason there was not often any marked rigidity of the limbs. In Case 10, however, rigidity was extreme and universal for some weeks, and in this patient many more patches of lymph were found scattered over the vertex, and especially over the Rolandic areas, than in any of the others.

At a comparatively early stage, many days or even some weeks before death, the infants usually passed through a condition of stupor into complete coma, and had to be fed entirely through the nasal tube; this was probably attributable to the increasing intra-cranial pressure, for the fontanelle became in every case tense or actually bulging. The only case in which there was no stupor was No. 11, in whom the symptoms were in many respects aberrant, and the pathological changes correspondingly slight.

The breathing was often irregular, sighing, or markedly Cheyne-Stokes in character; the pulse on the other hand was rapid and regular, or only showed such rhythmic irregularity as might be accounted for by the great respiratory variations. Although some of the cases were under observation from a very early stage I never at any time met with the marked irregularity, and still less with the slowing of the pulse, so frequent in and so characteristic of tubercular meningitis.

Vomiting not only occurred early, but was frequent throughout the course of the disease, and constituted an important symptom in every case. Constipation was never present, on the contrary rather diarrhoea, but this may fairly be regarded as a purely accidental complication arising in young infants breathing hospital air, often treated by mercurials, and necessarily fed by hand after having been as a rule previously suckled. The same considerations may also to some extent discount the significance of the sickness, at least in the later stages.

Squint was a common symptom; nystagmus was only noticed in two instances (2 and 5). Mr. Spicer examined the eyes in most of the cases, but only found distinct optic neuritis in two (7 and 10), in one (7) a very few hours before death; in a third case (6) the discs are described as blurred. In none of Dr. Barlow's cases was optic neuritis noted.

Towards the last rapid emaciation occurred, and in all but two cases was considerable or extreme at death, which seemed frequently to result from sheer inanition from the vomiting and the difficulty in getting food swallowed.

The temperature was normal throughout in every patient, except for an occasional rise to 101° or 102° , probably of no special significance; but, as in the tubercular disease, hyperpyrexia was a frequent precursor of the end.

In the majority of cases, both mine and Dr. Barlow's, the disease terminated fatally in from five to eight weeks; but in some the course was considerably longer, extending over some months, and it seems quite possible that all

active mischief may subside and a condition of ordinary hydrocephalus be left; this question, however, can more conveniently be discussed later.

Pathological anatomy.—In nearly every case the lesion found was a very definite one, viz. effusion of lymph, thickening of the pia arachnoid, and matting together of parts over the posterior and central area of the base of the brain, from the lower end of the medulla to the optic commissure; on the under surface of the cerebellum the thickening usually ended in a rather sharply defined margin half to three-quarters of an inch on either side of the middle line. The thickening and adhesions in the Sylvian fissures, usually so marked a feature in tubercular meningitis, were in these cases either entirely absent or when present only very slight in degree. The fold of pia mater extending between the medulla and cerebellum was usually much thickened, and in several cases, though certainly not in all, the cerebral ventricles were thus completely shut off from the spinal sub-arachnoid space.

Whilst the distribution of the morbid changes was very similar in all the cases, though more extensive in some than in others, the actual appearances varied considerably; in some there was a thick layer of puriform lymph, in some a thin layer only, and in others merely thickening of the pia mater, any lymph which might have been present having probably undergone organisation into fibrous tissue.

Isolated patches of lymph were noted in nine of the cases on the tip of one or usually both temporo-sphenoidal lobes (in the first case their condition is not recorded). In two cases of general purulent meningitis in infants I have found the lymph notably abundant in this position, but I have not observed it in cases of tubercular meningitis. In three of the cases (3, 4, and 11) patches of lymph were also present on the tips of the frontal lobes. Small isolated patches were also found occasionally on the vertex, but showed no regularity in their occurrence or uniformity in their distribution.

In all the cases there was well-marked distension of the lateral and third ventricles, and generally of the fourth also, by fluid varying in quantity from a few ounces up to half a pint or more, with corresponding flattening of the convolutions.

I regret that the spinal cord was not carefully examined in more of the cases; in none did the lymph appear to extend below the medulla, and although Dr. Barlow suspected the presence of spinal meningitis in two of his cases, the spinal canal was not actually opened. In several cases in which I examined the cord, it was firm and looked quite normal; in others, parts were very soft, but possibly this was due merely to absorption of an excess of fluid in the spinal canal after death, just as in cases of hydrocephalus the excessive quantity of fluid may be one cause of the frequently almost diffuent condition of the brain. In one case (8) examined only two hours after death, the cord was firm and appeared perfectly healthy.

In no instance was there any sign of tubercle either in the cranial cavity or in any other part of the body. In four cases the thickened membrane at the base of the brain was examined microscopically for tubercle, and always with a negative result. Although in some cases of tubercular meningitis, miliary tubercles may be found only in the cerebral membranes, a caseous focus can practically always be discovered somewhere in the body, most commonly in the glands; but despite careful search no trace of caseation was found in any of my cases, nor in Dr. Barlow's. The fluid from the ventricles was examined bacteriologically in only one instance (8), and was found to contain streptococci, but no tubercle bacilli.

Pathology.—The relation between the morbid process and the symptoms calls for no special discussion. The earlier symptoms—head retraction, vomiting, convulsions, &c., may be regarded as indications of cerebral irritation; the special incidence of the mischief upon the posterior part of the base of the brain, about the medulla and pons,

explaining the persistence and extreme character of the head retraction. Squint, when present, is most likely due to special implication of one of the oculo-motor nerves by the inflammatory process. The rigidity, twitchings, or slight convulsive attacks sometimes seen during the course of the disease, are in all probability the result of extension of the inflammation to the pia mater over the Rolandic areas. In the later stages the increasing stupor, passing into complete coma, with inability to swallow, Cheyne-Stokes breathing, and sometimes hyperpyrexia at the last, are doubtless due to the increasing intra-cranial pressure; that this stage frequently lasts so long may be owing to the almost exclusive occurrence of the disease in children so young as to have extremely yielding skulls.

The most important question, however, which is raised by these cases is the wide and difficult one of the origin of the hydrocephalus. Two possible causes have been frequently suggested and discussed, viz. an inflammatory effusion, or, blocking of the exits in the fold of pia mater closing the fourth ventricle, the so-called foramen of Majendie and other smaller apertures. This latter view seems at first a very plausible one, and was strongly urged by Hilton in his well-known 'Lectures on Rest and Pain,' in which he says: "In almost every case of internal hydrocephalus which I have examined after death, I found that the cerebro-spinal aperture was so blocked up—so completely closed—that no cerebro-spinal fluid could escape from the interior of the brain, and as the fluid was being constantly secreted it necessarily accumulated, and the occlusion formed, to my mind, the essential pathological element of internal hydrocephalus."

This theory, attractive though it sounds, involves, however, a number of suppositions in regard to the cerebro-spinal fluid which are very far from being proved; and there are, moreover, several very important arguments which tell directly against it. Firstly, there can be no doubt that in a large number—probably the great majority

—of cases of hydrocephalus, Hilton's statement to the contrary notwithstanding, the membranes at the base of the brain are perfectly normal; there is no need to cite individual cases to prove this.

Secondly, effusion into the ventricles occurs in cases of tubercular meningitis, in which, as shown very definitely by the investigations of Mr. Morton, of Bristol ('Brit. Med. Journ.,' 1893, vol. i, p. 741), there is certainly no closure of the cerebro-spinal aperture, and Dr. Wynter published four cases of tubercular meningitis, treated in the Middlesex Hospital, in which the ventricles were actually drained by opening the spinal dura mater in the lumbar region ('Lancet,' 1891, vol. i, p. 981). I have notes also of several cases of simple meningitis attended with considerable ventricular effusion, but without any thickening of the pia mater over the posterior part of the base of the brain.

Thirdly, it seems probable that even in posterior basic meningitis the opening is not closed in by any means all the cases. Thus, in Case 4 it seemed to be represented by a large circular aperture, surrounded by thickened membrane, though it is not possible to say with certainty that this was not accidentally produced in the removal of the brain; in Case 5, at the operation, fluid poured out immediately the dura mater was carefully opened, and at the autopsy there was no evidence of thickening of the membrane between the medulla and cerebellum; in Case 8 there was a small but distinct opening in the thickened membrane through which fluid passed freely; in Case 9 there was certainly no closure of the usual orifice, nor was there probably in Case 11; it was patent also in one of Dr. Barlow's cases.

Hydrocephalus sometimes supervenes upon epidemic cerebro-spinal meningitis, and has been attributed to blocking of the foramen of Majendie; but Dr. Merkel, of Nuremberg, has recorded a series of such cases in which a large quantity of fluid was also found beneath the spinal arachnoid around the cauda equina; this would, of

course, be obviously inexplicable on any mechanical theory. At most, therefore, obliteration of the foramen of Majendie can only be an explanation of the accumulation of fluid in the ventricles in some of the cases; and if we are dealing with a definite disease there ought surely to be one and only one definite cause for so important and constant a condition as the hydrocephalus.

Is the effusion then inflammatory? Two objections at once suggest themselves; firstly, neither the choroid plexuses nor the ependyma showed any naked-eye appearance of inflammatory change, although in three cases a few flakes of lymph were found in the effusion. Of course it may be argued that inflammatory changes in the ependyma may only be recognisable by the microscope. Secondly, the fluid has certainly not the characters of an inflammatory exudation; in the cases (5 and 6) in which it was obtained from the ventricles by tapping during life, its specific gravity varied between 1004 and 1006, and it contained only the merest trace of albumen; in two other cases (9 and 11), when obtained after death, it showed a very small quantity only of albumen, due probably to a little blood with which it unavoidably got mixed. In one instance (3) the fluid obtained at the autopsy thirty hours after death was highly albuminous, but I do not think much importance can be attached to this, for it is probable that the composition of the fluid rapidly alters when it remains in contact with the dead and softening brain tissue.

To escape the difficulty presented by the chemical composition of the fluid it has been suggested that an inflammatory effusion into the cerebral ventricles must have different characters from one elsewhere. Thus in Fagge and Pye-Smith's 'Medicine' (3rd edition, vol. i, p. 665), it says that "in one and the same case the various serous membranes may pour out fluids of very different specific gravity, and it is not improbable that the ventricles of the brain may continue to secrete a fluid containing scarcely any albumen, even when the process

is inflammatory ;” or, as it is put somewhat differently in Ziemssen’s ‘Cyclopædia,’ “some nutritive disturbances, as yet unknown, are undergone by the delicate vessels of the pia, in consequence of which a rapid transudation of fluid becomes possible.”

It is necessarily difficult to disprove such a theory directly, but it suggests rather an ingenious attempt, disguised in scientific phraseology, to suit the facts to the theory, rather than the theory to the facts ; at any rate there seems no doubt that under suitable conditions of sufficiently acute meningitis the fluid poured out does assume the characters of a normal inflammatory exudation. Dr. Halliburton (‘Chemical Physiology and Pathology,’ p. 356) says that “in cases of tubercular meningitis the specific gravity of hydrocephalic fluid rises and the solid constituents also increase, especially the proteids ; and in such cases the fluid resembles the exudations which occur in inflammations elsewhere.” Certainly in many cases of tubercular meningitis, the post-mortem appearances hardly suggest that the inflammatory process is so much more acute than in the simple basic form, as to lead to such a marked difference in the chemical composition of the resulting exudation.

There still remains a third explanation of the ventricular effusion, viz. that it is a simple dropsical exudation. Dr. Bastian, in a paper published by him in 1867, “On the Pathology of Tubercular Meningitis,” describes a case of that disease, with considerable effusion into the ventricles, associated with thrombosis of the veins of Galen ; the velum interpositum was much thickened, and the superficial veins of the ventricular surface were all distended. He attributes the effusion to venous hyperæmia, due to the obstruction in the veins of Galen. After my attention had been directed to this observation I examined the veins of Galen carefully in every case of posterior basic meningitis, but found thrombosis in one only (8), and in that it was evidently quite recent ; neither have I seen it in any case of tubercular meningitis, nor

have other observers (Dr. Gee and Mr. Morton) who have looked for it. Although, however, no cause for a dropsical effusion was found within the veins, yet in several instances I noted marked thickening of the fold of pia mater in the great transverse fissure, sufficient, probably, to cause very considerable obstruction to the flow of blood through the thin-walled veins there, and in Case 9, in which this fold of pia mater was not much thickened, the effusion was much smaller than in most of the other cases. Moreover, even before my attention had been called to Dr. Bastian's suggestion, I had been struck in more than one case by the marked fulness of the veins on the walls of the lateral ventricles, and subsequently I found this to be present in every case examined, so that in seven out of the ten necropsies, fulness, and generally very marked fulness, of these veins was noted, whilst in the remaining three the same condition may have been present, but there is no record, for at that time not being aware of its significance I was not on the look-out for it. The first time I was impressed by this venous fulness was in Case 5, which died a few hours after being trephined, from rapid escape of fluid, and I attributed it to the sudden fall of pressure within the ventricles, consequent on the withdrawal of fluid; but this explanation would not, of course, hold good for the other cases, and I can only attribute it to some obstruction to the return of blood, most probably in the veins of Galen, and an obstruction sufficient to produce such venous engorgement would seem to be adequate to lead to the effusion, and would also account for the œdematous condition of the brain substance found in nearly all the cases.

Nevertheless, although I am inclined to regard the hydrocephalus as mainly dropsical, it is quite possible that in some, at any rate, of the cases an inflammatory process may be contributory, and very probably the combination of the two, in even slight degrees, would be very efficacious in causing rapid effusion, in the same way as hepatic

cirrhosis is brought about very quickly by a combination of even slight alcoholic excess with congestion resulting from mitral disease.

I must not digress into any attempt to explain the excess of fluid often present in ordinary tubercular meningitis, but doubtless the same causes are operative. In some instances the fluid may be, as Dr. Halliburton's investigations indicate, inflammatory in origin; whether it can be so always seems doubtful. Thus the fluid drawn off by Dr. Wynter, in the cases already referred to, had a specific gravity of 1004 to 1006, and was almost non-albuminous. Several times, also, I have found very distinct thickening of the velum interpositum and of the pia mater in the great transverse fissure, with more or less dilatation of the veins on the ventricular walls; so that it seems probable that at least in part, and in some cases, the effusion may be dropsical in origin, or, in other words, that, as in the simple basic meningitis, both causes are contributory, though inflammation may play a more important part in the tubercular than in the non-tubercular disease.

This explanation of the hydrocephalus, as due partly to inflammation and partly to venous obstruction, has the advantage of accounting for its presence in all the different inflammatory conditions of the membranes with which it is associated, and in which there is no blocking of the cerebro-spinal aperture.

Meanwhile, the characters of the effusion in both acute and chronic hydrocephalus call for careful investigation at every opportunity.

Ætiology.—The disease seems to be almost confined to infants under one year old, and, as is to be expected at this period of life, to be about equally common in the two sexes, whereas tubercular meningitis is less common in infants than in older children, and more frequent in boys, perhaps from their greater liability to injuries and blows on the head.

It is noteworthy that in nine out of my eleven cases

the infants were still being fed from the breast only at the date of onset of the disease, one had been weaned at ten months, not many weeks before admission, and there is no note as to the remaining child; as would therefore be expected, in none was there any marked rachitic condition.

As to the actual cause of the disease, the question of injury is the first to demand consideration. In the first case convulsions and vomiting were distinctly said to have begun the day after a blow on the head, and Dr. Barlow obtained a similar history in some of his cases. What may be the exact connection between the two it is difficult to say: that an injury is *the* cause in all cases is inconceivable; that it may act as an efficient exciting cause, when other conditions are favorable, is possible. The position seems to be similar in regard to tubercular meningitis; obviously no injury could produce that disease in the absence of the tubercle bacillus, but possibly if the bacilli be circulating in the blood a blow on the head may determine their incidence upon the membranes of the brain, just as an injury to a joint may be the starting-point of tubercular arthritis; *e. g.* in a boy aged 3 years, in whom tubercular meningitis was fatal about six weeks after a severe blow on the head from a stone, at the autopsy I found miliary tubercles in the cranial cavity only, together with an old caseous focus in the right lung. In gouty conditions a similar relationship to injuries is frequently observed.

Ear disease must, of course, be thought of as a possible cause, but the evidence that it is so seems to me of the slightest. In seven of my cases both middle ears were healthy; in each of the other three inodorous pus or semi-purulent mucus was found on one side, but no other mischief whatever, and we must remember that it is exceedingly common in infants to find thick gelatinous mucus or mucopus in the middle ears; the readiness with which this, under the influence of a slight catarrh, may become purulent, probably explaining the frequency of otorrhœa in young

children. Of course, otitis media by spreading along lymphatic or venous channels may set up inflammation of the cerebral membranes, just as it may a cerebral abscess, without any disease of intervening parts ; but in such cases the resultant meningitis is probably always of a very acute character, and I have made autopsies upon several cases of acute purulent meningitis which have most likely been produced in this way.

Dr. Gee, in the ' St. Bartholomew's Hospital Reports ' for 1872 (vol. viii), described cases of purulent meningitis and otitis interna (unattended by any disease of the bone), and showed that the symptoms of the otitis may precede those of meningitis, may show themselves in its course, or be latent and undiscoverable throughout ; but, be it noted, these were all cases of purulent meningitis. Moreover, as will be explained directly, I do not consider any cause to be satisfactory, which will at most explain only a minority of the cases.

Congenital syphilis has, of course, been invoked as a cause of the disease, but there is, I think, an ever present danger of endeavouring, without sufficient reason, to make it responsible for this and for many other obscure conditions for which no other explanation is readily available. Any one sufficiently energetic in pushing inquiries can, in a large proportion of infants seen in hospital practice, elicit some facts in the history or in the child's condition which are capable of being attributed to syphilis, and when once some evidence of it has been obtained in a majority of the cases, it is very easy to argue that similar evidence would be forthcoming for the remainder, if only all the facts were available. Such a line of argument, though sufficiently tempting, is obviously open to many fallacies ; that syphilis may produce a localised meningitis is admitted, but there seems nothing to prove or even to indicate that it leads to the particular form of meningitis now under consideration. There was no strong evidence of syphilis in any of my cases, in most there was none at all. The mere occurrence from time to time of basic

meningitis in obviously syphilitic infants is not of much importance, since hereditary syphilis is certainly sufficiently common to render such a coincidence not infrequent; in fact, if the coincidence did not sometimes occur it might fairly be argued that the two diseases were antagonistic. That it can be the cause in some cases and not in others seems most unlikely, for I have endeavoured to show that we are dealing with a disease as definite as tubercular meningitis itself, a disease with well-defined and easily recognisable symptoms, running a pretty definite course, and presenting fairly constant pathological appearances; in fact, in all these respects, the variations met with are hardly if at all greater than those seen in different cases of tubercular meningitis.

It is, no doubt, possible for diverse causes to produce the same result, but I can hardly regard it as conceivable that this definite clinical and pathological condition can in some cases be due to syphilis, in some to traumatism, in some to middle ear disease, in some to other causes; for no one probably will contend that any one of these possible causes will explain all the cases. Therefore, instead of always thinking that, because syphilis is or may be a cause of so many different morbid conditions, we have done our duty by industriously striving in this or in other obscure diseases of infancy to ferret out a history or even the remotest possible suspicion of syphilitic taint, let us rather try and find some one cause which will serve as a sufficient explanation of all the cases.

If, then, we are led to think of posterior basic meningitis as a definite disease, with a definite—perhaps specific—cause, we are naturally also led to consider whether it has any relationship with that distinctly specific disease, acute cerebro-spinal meningitis. The latter occurs in epidemics which are certainly rare in this country, but it is in accordance with the analogy of many other diseases,—and the behaviour of cholera in Europe during the last few years may especially be referred to,—that sporadic cases, milder, more chronic, modified in character, and not liable to spread,

may from time to time occur; and most writers describe such cases.

The resemblance in the pathological anatomy and symptoms of the two conditions, allowing for differences in severity, acuteness, and duration, is somewhat marked. The epidemic meningitis is certainly common in young children; in fact, according to Dr. Oscar Medin, of Stockholm (quoted by Dr. Eustace Smith), infants under twelve months old are specially liable to it. Dr. Lewis Smith, of New York (Keating's 'Encyclopædia of the Diseases of Children,' vol. i, p. 514), states that "a larger proportion of fatal cases are in the first year of life than in any other year;" and Dr. Gowers says that "children possess a special liability, and in some epidemics have been almost exclusively affected."

The most marked lesion is purulent lymph, which may be present anywhere over the surface of the brain, but most abundantly and constantly at the base, especially at the posterior part over the medulla; it is found also along the posterior part of the spinal cord. There is effusion into the cerebral ventricles.

The onset of the disease is described as sudden, in children it is said often with convulsions; severe and often persistent vomiting, with extreme retraction of the head, are always marked symptoms, and opisthotonos is sometimes present. Twitchings of the limbs are very common, and general convulsions may occur during the course of the illness. Squint is frequently, and nystagmus sometimes seen. Optic neuritis was only found six times in forty cases. The pulse is usually quickened, and not often irregular or infrequent. The knee-jerks vary, sometimes being increased and sometimes absent. The temperature is very uncertain, but rarely high. There is great and rapid emaciation, and death—preceded by coma—is the usual termination in from two or three days to three or four weeks. The attacks vary much in severity, and abortive ones are described. In the epidemics a rash is often present, but is said to be absent in most sporadic

cases. Finally, as if to complete the resemblance to posterior basic meningitis, in cases of recovery chronic hydrocephalus has supervened, and post-mortem the cerebro-spinal aperture has been found to be closed.

On the other hand, it may be objected that nearly all forms of meningitis are commoner in children than in later life, and that a mere resemblance of symptoms in cases of intra-cranial disease is of no great importance, and may even be very misleading, in the absence of other indications of identity between what may be different diseases; for we might, in fact, draw as close a parallel between the simple meningitis I am describing and the tubercular form, as between the former and cerebro-spinal meningitis. Also it may be said that no such affection of the spinal cord has been made out as to justify us in regarding it as closely allied to the cerebro-spinal disease; although if the base of the brain be the part most affected in the ordinary cases of the epidemic variety, it may well be the only part attacked in milder sporadic ones.

On the whole, I think that clinical observation and even morbid anatomy will not carry us much further, especially in this country, where the epidemic form of meningitis is so rare, and that we must turn next to bacteriological investigation to help in determining the true pathology of the disease. That it is associated with a definite micro-organism is, I think, very probable, but on this point I am unfortunately unable to give any information. The bacteriology of the epidemic disease has not yet been satisfactorily worked out; but, so far as has been ascertained, the organism which is associated with it seems to be identical with, or closely related to that which is responsible for croupous pneumonia, and the pneumococcus is found also in most cases of acute purulent meningitis which have no obvious cause (Dr. Ormerod's article on "Epidemic Cerebro-spinal Meningitis" in Dr. Clifford Allbutt's 'System of Medicine,' vol. i, p. 673). It is, of course, possible that the pneumococcus, whilst giving rise to sporadic cases of meningitis, may, under

suitable conditions, cause an epidemic variety, just as it occasionally seems to give rise to an epidemic and infectious pneumonia; but we still know far too little of the part it plays in morbid processes generally, to be able to hazard more than the most vague hypotheses on these matters. Certainly in the four or five cases I have seen of meningitis in children associated with, and probably secondary to pneumonia, neither the symptoms nor the post-mortem appearances were in the least suggestive of the posterior basal form; the meningitis was more or less general, the lymph abundant, and, as a rule, the base of the brain, particularly the posterior part, was less affected than the vertex. Mr. D'Arcy Power ('The Clinical Journal,' vol. viii, No. 4) has noted that more children suffering from meningitis have been admitted to the Victoria Hospital since the epidemic prevalence of influenza during the last few years; but in the cases I have recorded no connection with this disease could be traced, nor did they occur specially during the actual outbreaks of the influenza.

Before we can determine that any micro-organism is the true cause of the posterior basic disease, much work will have to be done, not only in connection with this, but also with other obscure forms of meningitis, in order to clear up their mutual relationships; and even should a definite result be obtained, we should still be only on the threshold of the problem as to why such a limited area of the cerebral membranes is attacked, and why infants, usually previously healthy, are almost exclusively affected.

Some further help in regard to the relationships of the disease may be gained by the investigation of non-typical cases, especially by endeavouring to trace connecting links with acute purulent meningitis. I have examined a considerable number of examples of the latter in infants, but without definite results; the disease in most is general, but the base of the brain is sometimes not involved at all or less so than the vertex, and the children in whom it

occurs are either marasmic or already suffering from some definite disease, whereas in the simple basic form the previous good health and nutrition of the patients is rather striking.

The following case may possibly have been an acute variety of the posterior basic disease. A child aged 5 months was brought to the Hospital in a moribund state; the only history obtained was that he had been ill for three weeks with convulsions and vomiting, and had had head retraction throughout. At the autopsy the body was much emaciated. At the base of the brain was a dense mass of yellow lymph, most abundant between the medulla and cerebellum; it extended along the velum interpositum into the lateral ventricles and over the choroid plexuses. There was also a thin patch on the tip of the left temporo-sphenoidal lobe. The convolutions were flattened, and the lateral ventricles contained 2 or 3 ounces of turbid fluid, with many flakes of lymph. The veins on the ventricular walls were markedly dilated. The spinal cord and the other internal organs were healthy, and there was no sign of tubercle anywhere.

Another question which naturally arises, is whether the disease is really limited to infants, for with our present knowledge it is difficult to see why it should be, although eventually we may not find this age incidence to be any more remarkable than the limitation of the severe forms of summer diarrhoea and vomiting to young children. Dr. Newton Pitt records a case in a girl, aged 3 years ('Med. Times and Gazette,' 1885, vol. ii, p. 827). Personally I have no experience of the occurrence of the disease after infancy; the nearest approach to it which I have met with was in a boy, aged, however, only 2 years, admitted to hospital August 20th, 1891, with a history of vomiting, headache, and stupor, developing gradually after a fall on the head five weeks before. In hospital there was squint, irregular pulse, and a little head retraction; not much sickness after admission. Death, preceded by convulsions and hyperpyrexia, occurred on

September 3rd. At the autopsy there was a good deal of lymph over the posterior part of the base of the brain; the lateral ventricles were dilated, and contained several ounces of turbid fluid. No evidence of tubercle was found anywhere, although the resemblance to tubercular meningitis was in many respects very close, and the nature of the disease remains obscure.

Dr. Bastian kindly allows me to mention the following case, which was under his care in the National Hospital, Queen Square. Patient, a girl aged 10 years, was admitted April 2nd, 1894. Her illness had begun suddenly three weeks before with headache, followed in a few hours by a convulsive seizure; afterwards there was headache, some head retraction, delirium at times, but no vomiting. In hospital there was considerable head retraction; no paralysis; a good deal of pain in the back; some general hyperæsthesia; no optic neuritis; no loss of consciousness; pulse about 120; temperature 100° to 103.4° . On April 6th the skull was trephined; a large quantity of cerebro-spinal fluid escaped, and patient died on the table. At the autopsy the pia arachnoid at the base was found much thickened; the vessels on the pia were deeply injected on each side; the lateral ventricles were not much dilated; there was considerable excess of sub-arachnoid fluid in the spinal canal, with great engorgement of vessels on the surface of the cord. Although the membranes at the base of the brain were specially affected, I think that both clinically and pathologically the case suggests a subacute general cerebro-spinal meningitis rather than the special posterior basic variety. As to whether there may be any relationship between the two, we are—as I have already said—at present quite ignorant.

Diagnosis.—Until some experience of these cases has been obtained there can be no doubt that during life it is very easy to regard them as examples of tubercular meningitis. A young child is seen for the first time lying in an unconscious state, its head retracted, breathing

irregularly, and swallowing with difficulty ; the history is of a short illness, very probably ushered in by convulsions and attended by severe and persistent vomiting ; examination of the chest and abdomen reveals nothing definite. What more natural than that it should be looked upon as an ordinary case of tubercular meningitis in a late stage ? In my own cases what first aroused suspicion was the persistence of the condition just described, for when a child suffering from tubercular meningitis has reached the stage of stupor and rapid pulse, its remaining term of life is commonly to be reckoned by hours or at most by a few days ; but these simple cases last on day after day and even week after week, with but little variation, except that the head retraction becomes if possible more extreme and the coma more profound. Consequently when at last death did occur the pathological changes were looked for with special interest, and the suspicions which had arisen during life as to the unusual nature of the case, were confirmed by finding that the lesions were not those of the tuberculous disease. After an experience of a few such cases, one naturally began to look out for them during life and to endeavour to diagnose them as soon as possible, and thus the first important step was made towards an early diagnosis of the disease, viz. the recognition of the possibility of its occurrence, for naturally we rarely diagnose diseases which we are not expecting to meet with. Thus prepared, we may, I believe, make a fairly confident diagnosis of most cases of simple posterior basic meningitis some time before the fatal termination.

True it is that the symptomatology of tubercular meningitis is so varied and irregular that it may at first seem impossible to say that any symptoms of acute cerebral disease in a child may not be due to it, nevertheless I think experience shows us that the cases of posterior basic meningitis have a fairly characteristic clinical course, difficult to indicate in definite terms, but yet sufficient to say in dealing with a case, that the symptoms are not those, or have not the characteristic grouping of those

met with in any case, however aberrant, of tubercular meningitis.

The age of the patient does not afford much help in diagnosis; tubercular meningitis is certainly commoner after the first year of life, but a small number of cases do occur earlier, and all we can say is that whilst in children much over one year the simple variety of basic meningitis is excessively rare, during the first year of life either form may occur with perhaps about equal frequency.¹

Perhaps the most characteristic of all the symptoms of the simple basic meningitis is the head retraction, for though often present in the tubercular disease, it is, I should say, rarely so marked and never so persistent throughout the whole course of the illness as in the cases now under consideration. It may, of course, arise from other causes than meningitis; Dr. Money ('Treatment of Disease in Children,' p. 456) mentions cerebellar tumour, rheumatic affection of the muscles of the back of the neck, ear disease, tender cervical glands, abdominal disturbance, and other peripheral irritations; in several of the cases recorded by Dr. Gee it was probably due only to the muscular weakness associated with rickets, or to gastrointestinal disturbance, but in such, as in some of the other conditions mentioned by Dr. Money, the other signs of cerebral disease,—convulsions, coma, &c.—would of course be absent, nor is the retraction likely to be extreme or persistent. Once, however, I saw very marked head retraction lasting for a fortnight before death in a boy, aged seven years, suffering from typhoid fever; nothing was found post mortem to account for it, and one can only suppose it was reflex and due to abdominal disturbance. In another case, a boy aged nine months, an out-patient, suffering from bronchitis, broncho-pneumonia, diarrhoea and vomiting, there was well-marked head retraction for five or six weeks, the child had every appearance of

¹ During the time that my eleven cases of the non-tubercular disease occurred at the Victoria Hospital I find records of nine autopsies on children under one year suffering from ordinary tubercular meningitis.

tubercular disease, but got quite well, the head retraction passing off as the lungs cleared. This symptom seems also to occur occasionally in meningitis not affecting the base of the brain, for in a child aged eight months with acute purulent meningitis it was extreme for ten days, though at the autopsy there was no lymph over the pons, medulla, or cerebellum, and no distension of the ventricles. On the other hand, it was altogether absent in Case 11 of my series, but this was an anomalous one and probably impossible of diagnosis during life. In all of Dr. Barlow's cases it was well marked.

A slow and irregular pulse is well known to be a characteristic symptom of the intermediate stage of tubercular meningitis, whereas in the simple basic form, so far as my experience extends, the pulse is rapid throughout, irregularity is rare, and when present is very slight.

Some less marked differences may be referred to. Vomiting usually ceases in the later stages of tubercular meningitis, as the symptoms of increasing intra-cranial pressure become more marked; but in the simple form it often persists to the last. The bowels are usually costive in tubercular meningitis, at any rate in the early stages; in the non-tubercular disease I have never observed constipation, though Dr. Barlow notes its presence in two of his cases. The temperature is generally somewhat, though not often much raised in the tubercular disease, whilst in the simple form it is often quite normal for long periods; in both conditions, however, there is frequently a rapid ante-mortem rise.

Lastly, the duration of the symptoms confirms the suspicions to which their character has already probably given rise, for whereas if tubercular the case ends fatally in two or the very most three weeks from the onset of definite symptoms, the simple form—as judged from Dr. Barlow's and my own cases—seems to last at least four or five weeks, commonly about six, and often considerably longer.

It is not necessary to discuss here the diagnosis of the disease from non-cerebral diseases which may for a time

simulate it, *e. g.* gastro-intestinal disturbance, typhoid fever, cerebral symptoms due to reflex irritation or pyrexia, &c. The points of distinction are necessarily much the same as those between tubercular meningitis and the conditions just mentioned, and are sufficiently dealt with in the ordinary works on medicine and on diseases of children.

The method of lumbar puncture of the sub-arachnoid space, which has lately been introduced as a means of diagnosing meningitis from other conditions, would not be reliable in the posterior basic disease, for in those cases in which the foramen of Majendie is closed, and the communication between the cerebral ventricles and the sub-arachnoid space of the cord consequently cut off, no indication could be obtained by the puncture of the amount of the intra-cranial pressure, or of the character of the intra-cranial fluid.

Prognosis.—Although the disease is undoubtedly most serious, yet there is good reason for thinking that a small proportion of the cases may and do recover, at least partially, for in some hydrocephalus will be left. It seems to me that in Case 5 recovery had certainly occurred so far as the meningitis was concerned: the child had presented all the characters of well-marked basic meningitis, and definite remains of it were found post-mortem; but all symptoms, notably the head retraction, vomiting, and difficulty in swallowing, had disappeared; the child was gaining weight, becoming more lively and observant, and was in fact suffering simply from chronic hydrocephalus. There had, I believe, been thickening of the pia mater over the posterior part of the base of the brain, which had very largely disappeared, but would probably have been still recognisable there but for the disturbance of the parts in the operation, and the almost diffuent condition of the brain at the post-mortem.

I have notes of another case in which there had apparently been posterior basic meningitis which had not proved fatal. Edith S—, aged 3½ years, was admitted to the Hospital on April 30th, 1889. Not much history could

be obtained, but she had been born with a spina bifida, had never attempted to stand or bend the knees, and was backward in talking and in cleanliness. The spina bifida was healed, but the child had rigidly extended legs with talipes equinus. For this tenotomy was performed on May 13th; broncho-pneumonia developed shortly after, and the child died on June 2nd. At the autopsy the pia mater between the medulla and the cerebellum was found converted into a firm fibrous membrane, and the cerebro-spinal aperture closed; there was no lymph, the change being evidently of very old standing. The lateral ventricles and the aqueduct of Sylvius were much dilated, and contained several ounces of clear fluid; velum interpositum and choroid plexuses rather congested. No sign of tubercle in brain, though the thoracic and mesenteric glands were caseous. Right middle ear healthy, left contained a little muco-pus. The anterior fontanelle was widely open, and the frontal suture unclosed. The case was evidently one of hydrocephalus associated with chronic changes in the membranes at the base of the brain.

The difficulties in the way of proving the possibility of complete recovery are obvious, and must necessarily be almost insuperable, the more so because such cases, if they do occur, will naturally be of a milder type than the fatal ones, and will consequently present less definite and less recognisable features. I have, however, seen a few infants who got quite well after an illness the symptoms of which most closely resembled those due to posterior basic meningitis; the following is a good illustration. Edith G—, aged 9 months, admitted May 7th, 1895. First child; no previous illness except bronchitis; no evidence of syphilis or injury; had had otorrhœa at times. Fed on breast only. Illness began rather suddenly about three weeks before, with frequent vomiting and persistent and increasing head retraction; the bowels had been regular; the child had taken the breast badly, and had wasted. On admission patient swallowed badly and took very little notice, crying only when disturbed; had marked

though not extreme head retraction, with some opisthotonos; fontanelle distinctly bulged; no convulsions, rigidity, squint, or nystagmus. Pupils equal and of moderate size; pulse rapid and regular; breathing regular; knee-jerks and plantar reflexes brisk; very slight rachitic changes in bones. The child remained in this condition, with little alteration, for some weeks; the head retraction was constant, but varied in degree; no convulsions, but slight rigidity at times. For the first five weeks vomiting occurred usually several times a day; bowels rather loose; pulse rapid (150 to 160) and regular; no optic neuritis; temperature rose occasionally, at first as high as 102.6° , but remained normal after the first week. The only drugs given were iodide and bromide of potassium.

Improvement slowly occurred, the head retraction and bulging of the fontanelle diminished, the vomiting gradually ceased, the child began to look brighter and became more natural in manner, and was discharged on July 17th. On July 23rd she was doing well, took notice, and was not sick; there was still slight head retraction. So far, at any rate, as symptoms can guide us, this case presented a fairly typical picture of posterior basic meningitis.

Two further questions arise; the first is as to whether some of the supposed cases of tubercular meningitis which recover are not really examples of this simple posterior basic disease. There is evidence, not necessary to adduce here, which seems to show that tubercular meningitis is perhaps not invariably fatal; but without entering upon this still disputable question, the cases of recovery must necessarily be excessively rare, especially when we consider that the disease is, as a rule, merely a part of a general tuberculosis. Simple posterior basic meningitis is a condition not as yet generally described or recognised, and, as has been shown, very easy to be confused with the tubercular form by those unfamiliar with it, and we may therefore, I think, fairly assume that, at any rate in infants, some at least of the credited recoveries from the latter were really examples of the somewhat less fatal simple form. That

such a child should afterwards fall a victim to tubercular disease, and even to tubercular meningitis, is certainly not impossible, and does not in the least prove, as has sometimes been assumed, that the first cerebral attack, old evidences of which may be found post-mortem, was necessarily of a tubercular nature.

The second question is, to what extent is this form of meningitis a cause of ordinary chronic hydrocephalus? One such case has just been mentioned, that of hydrocephalus and spina bifida, and Case 5, which was operated on, may fairly be considered another. Dr. Baxter recorded one in the 'Transactions' of the Pathological Society, vol. xxxiii; a child previously healthy had an attack of convulsions when six weeks old; at three months the head began to enlarge, and great hydrocephalus developed; death occurred at two and a half years. At the autopsy five pints of fluid were found in the brain, the cerebro-spinal aperture was obliterated, and there was also some recent meningitis over the pons and within the circle of Willis; no tubercle. Hilton, as already quoted, attributed most cases of internal hydrocephalus to closure of the cerebro-spinal aperture, and details several examples. The number of such cases must, however, I think, be few, for posterior basic meningitis is far from common, and of the cases which do occur probably most are soon fatal. Dr. Baxter himself said that though he had been looking for this condition in hydrocephalus, the one he described was the first he had found after examining about six cases. Moreover, non-congenital hydrocephalus begins, as a rule, with a gradual enlargement of the head, without any very marked disturbance of the general health—very different to the severe symptoms which accompany basic meningitis. Nevertheless, the possibility of the occurrence should not be overlooked, and in old-standing cases too much reliance must not be placed upon the absence of extensive pathological changes at the autopsy. Speaking of epidemic meningitis, Dr. Ormerod (*loc. cit.*, p. 668) says, "It would appear that in cases which survive the acute stages of the

disease, the meningeal exudation gradually degenerates and is absorbed, but the pia arachnoid becomes thickened and shrunken, and the *ependyma ventriculorum* hypertrophies. The ventricular effusion, probably in consequence of these cicatricial changes, either remains unabsorbed or increases in amount, albeit now less purulent and more passive in character." These words doubtless apply equally to those cases of posterior basic meningitis which are followed by chronic hydrocephalus, the more marked signs of inflammation subsiding, and only slight chronic changes being left; these may very easily be missed at an autopsy, for in advanced cases of hydrocephalus a satisfactory examination of the membranes at the base of the brain is almost impossible. Certainly Case 5, already referred to, suggests that a basic meningitis may occur, nearly all pathological evidence of which may, like the symptoms, disappear; and yet that it may start a ventricular effusion which may increase and become permanent.

Treatment.—As we must recognise the possibility, small though it be, of the inflammation subsiding and recovery taking place, we may justly, I think, employ some such vigorous line of counter-irritation as Hensch recommends for meningitis; not, perhaps, in such young children, leeches or blisters, but at least mercurial ointment to the throat and neck, an ice-bag to the head, and calomel internally. Most of the cases described were treated with iodide and bromide of potassium, or with grey powder or calomel, without, however, any obvious effect, except that of causing diarrhoea. Should there be signs of commencing hydrocephalus, the tense bulging fontanelle almost invites puncture of the lateral ventricles with a small trocar and cannula; and whilst this seems a harmless procedure, if carried out with proper precautions, it must surely be beneficial, should the meningitis be recovered from, by preventing injurious and permanently harmful pressure being exercised on the brain. Tapping does not, of course, prevent the supervention of chronic hydrocephalus, still there seems no reason why in some cases

the ventricular effusion should not be transitory, and in such, timely withdrawal of fluid during the acute stage may make all the difference between recovery with a useful or with a permanently damaged brain. Care must obviously be exercised, especially in earlier punctures, to remove only a small quantity of fluid at once, but my limited experience tends to show that in the soft compressible skulls of young children several ounces may be withdrawn without any harm resulting. Some writers say that with each successive tapping the fluid becomes more inflammatory in character, but in the two cases (5 and 6) recorded above, in which it was several times repeated, there was no such change.

Finally, the question of operative interference has to be considered; this may be resorted to either in the acute stage, as soon as marked pressure symptoms develop, as in Case 8; or later on, if, after the acute symptoms have subsided, the hydrocephalus is evidently increasing, in spite of the drawing off of fluid by trocar and cannula, as in Case 5. If the accumulation of fluid in the ventricles be due to closure of the cerebro-spinal aperture, it seems a reasonable thing to try and reopen it; whilst if it be dropsical in origin and produced by pressure on the veins of Galen, or even if it be due to chronic changes in the ependyma, continuous drainage from the sub-cerebellar arachnoid space may allow time for the inflammatory changes, either in the velum interpositum or in the lining membrane of the ventricles, to subside, and so permit of ultimate recovery. In either stage, acute or chronic, the most immediate danger in operating seems to arise from the difficulty in preventing too rapid an escape of fluid. The child, aged 10 years, under Dr. Bastian's care, to whom reference has already been made, was trephined over the right occipital region, between three and four weeks after the commencement of her illness, but died on the table from rapid escape of fluid; the same cause led to a rapidly fatal result in my Case 5, trephined long after all acute symptoms had quite subsided. Mr. Lowson, of

Hull, also records a case of hydrocephalus in which drainage of the fourth ventricle proved fatal in a similar way in a few hours ('Brit. Med. Journ.,' 1893, vol. i, p. 1322).

A few cases, however, have been recorded, which show that this is not an insuperable difficulty. In 1893 Mr. Parkin, of Hull, published the case of a child, aged 11 months, suffering from chronic hydrocephalus, in which he trephined below the superior curved line of the occiput, and inserted a horse-hair drain below the cerebellum into the subarachnoid space; fluid escaped slowly for fifteen days, and the drain was removed after eighteen days. The child was discharged greatly improved, and three months afterwards there was no indication of recurrence ('Lancet,' 1893, vol. ii, p. 1244). Mr. Parkin has since published a second successful case of trephining in a child of 3½ years, and also one in an infant aged 5 months, in which death occurred from hyperpyrexia on the ninth day from too rapid withdrawal of fluid ('Lancet,' 1895, vol. ii, p. 1166).

Dr. Glynn and Mr. Thomas, of Liverpool ('Lancet,' 1895, vol. ii, p. 1106), have also recorded a case of hydrocephalus in a man aged 18 years. The skull was trephined through the cerebellar fossa and the fourth ventricle opened up, after breaking through a soft partition, which they took to be the thickened posterior medullary velum bounding the fourth ventricle; a quantity of fluid escaped, and the wound was closed without any drainage into the cranial cavity. The patient recovered. All the above were chronic cases, but Dr. Ord and Mr. Waterhouse ('Lancet,' 1894, vol. i, p. 597) drained the subarachnoid space for eighteen days in what appeared to be a case of tuberculous meningitis in a child aged 5 years, with perfect success; so that the presence of recent inflammation constitutes no bar to the success of the operation.

True it is that probably none of the successful cases here mentioned were examples of simple posterior basic meningitis; but the conditions and indications for opera-

tion must be the same, and the chances of success at least as good in this as in the other varieties of meningitis, or as in cases of hydrocephalus due to other causes. The further discussion of this method of treatment I must, however, leave to the surgeons, although both from theoretical considerations and from practical experience I am inclined to think that operative interference offers at present but little prospect of success, and the argument sometimes advanced that because a case* will almost certainly prove fatal if left alone, it should therefore be operated upon—however remote the prospect of success—would, if frequently acted upon, and with almost uniformly fatal results, tend to bring surgical practice into disrepute.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 114.)

ON THE RELATIONS
BETWEEN
BODILY DEVELOPMENT, NUTRITION,
AND BRAIN CONDITIONS
IN THEIR PATHOLOGICAL ASPECTS

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THE purpose of this paper is to show, as the result of extended observation among children, that—

(1) Points of abnormal development in the body are very common, and have important signification.

(2) Abnormal nerve-signs, or points in balance and movement, have been defined; they are much associated with defective development, as well as with the causes of mental dulness, and are of clinical importance.

(3) There is an intimate empirical relation between
(A) Defective development; (B) Abnormal nerve-signs;
(C) Low nutrition and health of the body and its tissues;
(D) Mental dulness and backwardness.

(4) Some explanation of the relations among the classes of defect may be offered on the hypothesis of evolution (and dissolution of co-ordination) resulting from action of the environment.

Certain propositions will be put forward, supported by evidence accumulated.

Experience gained among children shows many types and groups of cases of clinical significance. All observers are familiar with ill-made heads and features ; delicacy without disease ; nervousness and nerve disorderliness without known structural changes ; children with antics, abnormal gestures, awkward habits in balance of body, in movement, in speech ; children mentally dull or backward ; and cases of varied grades of cerebral deficiency. It is always useful to study common conditions such as are frequently met with, and by careful accumulation and arrangement of facts some problems of importance in pathology may be explained.

The facts concerning conditions of bodily development below the normal, and indications of defective nerve status as so commonly seen, supply data for the more accurate study of what is sometimes called the constitutional or general condition of the subject examined ; while their fuller appreciation appears to throw considerable light upon such problems as the causes of infant mortality, the differences between the sexes, the genesis of neurotic conditions, feeble health without disease, and the many causes of mental dulness and deficiency.

In a paper read before this Society in 1882, "On Postures of the Hand as Indications of the Condition of the Brain," I put forward certain principles for the anatomical analysis of the typical postures described, and the usefulness of such points as clinical signs was discussed. After further observation and study they were applied in an inquiry as to the mental and physical conditions of children in schools ; their signification has since been further determined.

In 1888 a Committee of the British Medical Association

was formed to conduct an investigation as to the average development and condition of brain power among the children in primary schools ; and between 1888-94 I had the opportunity of examining 100,000 children individually in schools, in conjunction with other medical men. My thanks are particularly due to Dr. G. E. Shuttleworth, Dr. Fletcher Beach, Dr. T. L. Rogers, Dr. Henry Rayner, and the late Dr. D. H. Tuke, for their kind assistance.

The children were examined as they stood in rank, each being viewed systematically ; they were required to make a few simple movements, and all points below the normal both in body and in action were noted. The same points were looked for in each child. The teacher's report as to mental ability was recorded in each case. In this manner a written account in schedules was taken of 18,127 cases in some respect below normal. The results of observation have been published in a report :¹ the 104 signs described in the cases have been defined in a nomenclature ; while the cases presenting the classes of defect are analysed and arranged so as to show their relations to one another for the sexes in age groups.

Four main classes of defect were thus recorded :

(A) Defect in development : in size, form, proportions of parts of the body.

(B) Abnormal nerve-signs : abnormal balance and action, or reaction in movement and co-ordination.

(C) Low nutrition : cases pale, thin or delicate.

(D) Cases dull or backward mentally, as reported by the teachers.

Taking these records as a basis of facts, analysis and research has supplied much information as to the relations between bodily development, nutrition of body, and brain conditions in their pathological aspects ; as found among boys and girls respectively in the age groups.

¹ ' Report on the Scientific Study of the Mental and Physical Conditions of Childhood, with particular reference to children of defective constitution ; and with recommendations as to education and training. Based on the examination of 100,000 children, 1888-94.' Published by the Committee, Parkes' Museum, Margaret Street, London, W.

The more common points observed as defects in bodily development numbered forty-two, besides miscellaneous examples, such as congenital defects of the eyes and the grosser deformities of the body. The principal signs are in size, form, and proportions of the cranium, palate, external ear, and the features individually, as well as in stature and growth. The relative importance of each defect varies; the association with other classes of defect standing highest where the cranium is ill-made, the palate standing next in value. These individual co-relations are given in Table XVI of the report.

When two or more developmental defects are present in the same case, the co-relation with other classes of defect rises (see Table XVIII in Report).

The more common abnormal nerve-signs described numbered twenty, besides such less common defects as tremor, ptosis, and other paralyses. These signs are abnormal modes of muscular balance, movement and reaction in movement; they are seen in the head, spine, shoulders, and planting of the feet and balance of the upper extremities; while as finer movements we observe the nerve-muscular balance and movements of the hands and digits, movement of the eyes, together with the expression, muscular tone, and movement in the frontal middle region and lower part of the face.

In dealing with a large number of cases facts must necessarily be recorded by the aid of figures. In the report published and elsewhere,¹ results of statistical analysis are fully given; they form a basis of facts here quoted, and need not be repeated. In all such modes of dealing with accumulated cases the boys and girls are kept separate. Another important point as regards the 50,000 children seen, 1892-4, is the method of dealing in statistics with "primary groups of cases;" that is, with groups in which observation showed the presence of the class of defect named, and absence of the other classes of

¹ See author's paper, 'Journal of Royal Statistical Society,' March, 1893, and March, 1896; Reports to British Association, 1894-6.

defect. This has made it possible to trace with some degree of accuracy the interaction of the various forms of defect as referred to in evidence for the propositions. Cases are arranged not only in primary groups as above; they are also presented as "compound groups," containing all the cases with the class or classes of defect named. Thus Primary Group A contains all cases with defect in development only without nerve-signs or low nutrition; while the Compound Group A¹ contains all the cases with defect in development, whether they have also nerve-signs and low nutrition or not. These compound groups are necessarily larger than the corresponding primary group. The facts derived from the results of the inquiry are sufficiently shown in the tables appended; they deal only with the 50,000 children seen, 1892-4, giving an analysis of all cases with developmental defect; abnormal nerve-signs or low nutrition, whether dull pupils or not. The facts concerning mental dulness are here omitted.

Table I shows the primary group, or groups presenting only the class or classes of defect named, presented as sub-classes comprising the total number of cases noted. Cases are arranged in three columns as age groups; and in the fourth column for children of all ages. In the first four columns the primary groups are distributed as percentages of the total number of cases noted respectively; in the last column the distribution is in percentages upon the total number of children seen.

Table II shows the compound groups, containing all the cases of the class or classes of defect named, whether alone or in combination with other defect.

The groups are presented in columns as in Table I, and the percentages are similarly distributed.

Table III shows the co-relations of the classes of defect or the association of the groups of cases with other class or classes of defect named, in the form of percentages, whether alone or in combination with other defect.

Table IV shows the primary groups as components of

¹ Ibid.

the compound groups which contain all the cases with the class of defect named. The number of cases is given ; while the primary groups are distributed as percentages on the number in the compound groups respectively.

In explanation of the tables the information given as to the associations of developmental defect, for children of all ages, may in part be traced. In Table I it is shown that such cases alone, without either nerve-signs or low nutrition, formed 26·9 per cent. of the boys and 17·5 per cent. of the girls noted as cases with some defect, and 4·5 per cent. of the boys and 3·2 per cent. of the girls seen in schools. In Table II it is shown that developmental cases alone or in combination with nerve-signs and low nutrition formed 26·9 per cent. of the boys and 23·6 per cent. of the girls noted with some defect ; and 8·7 per cent. of the boys and 6·8 per cent. of the girls seen in schools. In Table III it is shown that of the developmental cases 38·4 per cent. of the boys and 36·3 per cent. of the girls respectively presented abnormal nerve-signs ; while of the boys 16·2 per cent., and of the girls 26·5 per cent., were pale, thin, or delicate.

In Table IV the numbers of boys and girls with developmental defects only, and the total numbers with such class of defect, are shown ; while the percentages show the proportions of the primary group to the compound group.

In studying these records of the common defects of childhood the statistical method shows the existence of important relations among the classes of defect ; and shows further, that a clinical value may be attached to the signs by which the groups of cases are classified. Arguments in explanation of the co-relations demonstrated may afford some explanation of the significance of the developmental defects and abnormal nerve-signs. In studying the physiology of man, and the pathology of his parts or organs, we consider growth, function, change and decay in the part or organ and in the cells composing its structure (cellular pathology). So in studying the

brain we wish to observe function and change in its parts—the nerve-centres, cells, and fibres. I wish here to suggest the importance of noting the attributes time and quantity of the growth, or action, in the living part observed, as part of the natural phenomena recorded.¹

Analogy or comparison between objects or phenomena is one of the means of giving explanation, and is admissible in scientific argument if the points of comparison as observed are accurately described. What common points for comparison are to be found between examples of mal-development or disproportioning in the growth of the parts of the body and abnormal modes of action in the brain-centres? A careful analysis of the signs observed in defective development and the abnormal nerve-signs shows that we may logically compare relations in proportional growth with relations in quantity of nerve-action; and relations in the time of growth with relations in the time of action in nerve-centres.

The signs of defective development can be shown to consist mainly of disproportion in growth, the ratios of growth in the parts of the features are not normal; this is a relation in the quantities of growth, abnormal under the action of inherited or present effects of the environment. As an example, the external ear may be large, outstanding, coarse-looking. Analysing the parts of such an ear we usually find that growth has been greater on the posterior than on the anterior surface, and the outstanding ear is a mechanical result of such proportions in growth; further, the ante-helix is absent, and the helix in great part is wanting, another abnormality in the proportions of growth. The ratios of growth in the parts of the ear are less varied, and not the normal. Such an outstanding ear is probably a better sound collector than the normal ear; at any rate sound waves are better collected in a normal ear by

¹ See author's 'Physical Expression,' International Scientific Series. 'Anatomy of Movement: a Treatise on the Action of Nerve-centres and Modes of Growth.'

holding it forward and mechanically unfolding the antehelix. The normal ear cannot be said to have evolved in its mode of growth to produce better hearing, but is the result of complex ratios in growth. These abnormal external ears are far more common in boys than in girls; the male always tends more to variation in bodily development. Of cases with abnormal ears 54·0 per cent. of the boys and 47·7 per cent. of the girls presented abnormal nerve-signs.

The physical signs indicating the mode of action among the nerve-centres express relations as to time and quantity of action. In a normal child the balance or posture assumed by the hands when held out free in front of him to the word of command, or in imitation of the action in the observer, is with the forearm pronate, the arms being straight at the elbow, horizontal on a level with the shoulder and parallel to one another the width of the chest apart, while the wrist is straight extended, and all parts of the hand are balanced in the same plane, both the metacarpal bones and the digits. Such was the type of hand balance seen in 94·8 per cent. of the boys and 95·4 per cent. of the girls in the schools.

In a paralysed arm without rigidity, or, better, in a healthy arm during deep sleep, when the brain is not innervating the muscles, if the forearm be held passively horizontal it will droop at the wrist, the metacarpal bones folded together or adducted while the digits are moderately flexed. In the child just convalescent from chorea the hand when held out droops in flexion, the metacarpus being slightly adducted while the metacarpo-phalangeal joints are extended backwards beyond the straight line, the fingers being slightly flexed. In this nervous hand posture there is a difference of balance both from the normal straight hand and the adynamic hand of paresis; we infer altered ratios of action among the nerve-centres. The centres for wrist extension are weakened while there is over-action in the centres for the small muscles extending the knuckles; there is an altered ratio of action

indicated by the posture, and those ratios are not normal. Unequal bilateral cerebral action is indicated when the head habitually drops to one side, and when the left arm is held at a lower level than the right, with the hand more in the adynamic posture, as is very commonly seen in weak subjects.

Twitching of individual fingers, whether flexor-extensor as produced by the muscles of the forearm, or lateral as produced by the smaller intrinsic muscles of the hand, are indications of temporary and often uncontrolled discharge in the nerve-centres expressing their frequency and rapidity, or other relations in time of action.

Co-ordination consists in the regulation of the attributes time and quantity of the individual movements of an action; and consequently in control of these attributes in the motor action of the nerve-centres in certain combinations and series of combinations.

Movements may be conveniently classified for clinical observation as series of acts, according to their relations in time, thus—

1. *Uniformly repeated series of movements.* The same parts moving in similar combinations and series of acts on successive occasions, often recurring on any stimulation through the senses. Over-action of the frontal muscles in children of cerebral deficiency is a type of such mode of movement, producing horizontal creases in the forehead which may be coarse or fine but are constantly repeated; the frontal muscles are sometimes seen working under the skin in vermicular fashion with an athetoid movement. Repeated grinning is an example of an action of this class; while in the athetosis of hemiplegia we see uniformly repeated series of movements uncontrolled by impressions through the senses, and therefore useless and purposeless.

2. *Augmenting series of movements.* The area of movement spreading from part to part either spontaneously or on any stimulation; the number of parts in movement increasing; such mode of action in a normal brain may

be indicative of mental excitement or emotion. The area of parts moving may spread in the face, then to the digits of one hand or both hands, just as the march of spasm is seen in epilepsy and with some cerebral lesions. Such augmenting series of movements are often seen on approaching a choreic child, especially when he is spoken to. In other cases an extra series of uniformly repeated movements may accompany co-ordinated action, as when the child writes his name, the action being accompanied by over-action of the frontal and corrugator muscles. The question asked, or the commencement of any (voluntary) action may be accompanied by protrusion of the tongue, bending of the head to one side, fidgeting extra and uncontrolled movements of the fingers; analogous extra movements may occur in wandering of the eyes.

3. *Diminishing series of movements.* A diminishing area of movements, and number of movements, corresponding to a diminishing number of nerve-centres in action, is seen as sleep approaches, or excitement or a storm of passion subsides; as the epileptic attack passes off; or as we watch a child day by day when recovering from chorea.

4. *Co-ordinated movements.* A series of movements controlled or adapted by impressions received. In a clinical examination the child may be asked to imitate movements with one or with both hands as made before him; he does so through the agency of sight. If his movements, as controlled through his eye, are the same as those imitated, alike in the time and the quantity of movements of parts, he is said to perform good co-ordinated movements.

The only characters of a movement are its attributes time and quantity, together with its antecedents and sequents.

In what respects, then, does a child with abnormal nerve-signs show abnormal brain action? The four classes of movements are described as to their relations in the attributes time and quantity of action; the same attributes as characterise modes of growth in parts of

the body as normal or abnormal. Let us take some examples. Response to a question or in imitation of movement may be delayed, then we see the action slow or abnormal in relation of time. In uniformly repeated movements, and in the occurrence of extra movements, abnormality in action of the nerve-centres is in relation of time. In abnormal balance of the hand we see the ratios, or relations in quantity of action in the nerve-centres respectively, indicating the abnormal or imperfect nerve status; this may affect centres for the larger or the smaller muscles.

The inference from the observed co-relation of development defect with the nerve-signs is that when the proportions in growth of parts of the body are abnormal (relations in quantity), whether as the result of inheritance or of impressions received, the nerve-centres are commonly so built up that in subsequent action they do not work, under the influence of the environment, in normal relations of time and quantity. This defective mode of action among nerve-centres may apply equally to motor and mental function.

Following the lines of the hypothesis of an evolution of living beings under the action of their environment, an attempt may be made to analyse, or in part unravel, some factors in the ætiology of irregular bodily development and nerve status.

It has been shown that the defects in development are disproportioning in parts of the body which is mainly a relation in the ratios of growth or their delay or arrest; while the abnormal nerve-signs are the expression of abnormal relations in the quantity or the time of action in the nerve-centres producing the balance or movement seen.

Each class of signs is then essentially due to defective relations in time or quantity of action as controlled by the environment; it has been shown that the two classes of defect are co-related, and when so associated in a persistent form they are apparently dependent upon, or

sequential to, circumstances which affected both the ratios of development in parts of the body and the building up of the nerve-centres.

It has been shown that defects in bodily development are much associated with conditions of low nutrition; such children being pale, thin, or delicate. This fact seems to indicate that such environment as leads to disproportioning in growth leads also to a status of the tissues prone to atrophy under adverse circumstances. This, I know not why, is specially found among girls, and acts very unfavorably on their neural conditions; hence the great liability among girls of mal-development to acquire nerve disturbance taking a more permanent form than among boys.

Inco-ordinated nerve action without structural changes, is mainly a state in which the time and the quantity of action in nerve-centres is abnormal as it occurs under control (or want of control) of the environment. In some cases, as in child-fidgetiness over spontaneous action, and in chorea, it appears that the mode of action among the nerve-centres reverts to the spontaneity and the inco-ordinated condition of brain which is normal in the infant, as evidenced by the large number of spontaneous movements of all parts which characterises the healthy child during the early months of life (microkenesis¹).

Further facts in evidence are given in the following propositions.

PROPOSITION I.—*Disproportioned bodily development is very common; it is co-related with low nutrition of body and inco-ordinated brain action.*

The facts concerning the association of mal-development with low nutrition of body are demonstrated in the tables, and in the explanation of them already given. This co-relation is highest (see Table III) with the girls in all age groups, and is specially marked among the children

¹ See 'Proceedings of the Royal Society,' vol. xlv, and 'Journal of Mental Science,' April, 1888.

of seven years and under ; this co-relation is much lower at eleven years and older. On the other hand, the co-relation of developmental defect with nerve-signs increases with age for girls, less so with boys. Finally, the proportion of the developmental cases that have not acquired either low nutrition or nerve-signs is highest for boys in the oldest group ; while for girls it remains about the same in each age group (see Table IV). This seems an indication that the environment is apt to produce more harm and less good in girls than in boys of imperfect development.

PROPOSITION II.—Inco-ordinated brain action is much associated with mal-development ; and the presence of nerve-signs is more co-related with the causes of mental dulness than are the signs of defective development.

The tables show the co-relation of nerve-signs with mal-development (Table III) as highest for children of seven years and under, and lowest in the oldest groups ; while the proportion of nerve cases that have nerve-signs only without developmental defect or low nutrition (Table IV) is highest with the oldest group ; the environment appears to favour the occurrence of simple cases of nerve inco-ordination and weakness ; these in many instances might be removed by training. Although the facts concerning mental dulness are not included in the tables here given, it may be stated that the presence of abnormal nerve-signs, indicating inco-ordination of brain, is more closely related to the causes of mental dulness than are the defects in bodily development.¹

PROPOSITION III.—Delicate children are in a large proportion of cases ill-developed in some part of the body.

The relation between delicacy, as indicated by the child being pale or thin, and mal-development is not as strongly marked in boys as in girls, and the co-relation varies in the age groups. Among children without developmental

¹ See 'Mental Faculty,' Cambridge University Press.

defect there does not seem to be a larger proportion of delicate girls than boys ; it is the girl who has some defect that is delicate, not all girls. In this co-relation there is very little difference whether nerve-signs are present or not ; I conclude, therefore, that the low nutrition in developmental cases is not a direct effect of the accompanying brain disorderliness. It is interesting to find that ill-developed children remain thin and delicate in all social classes and among those well supplied with food ; in resident institutions, where there is but little mental excitement, the associated delicacy is least marked.

TABLE II.—For 50,000 children seen 1892-94. Showing the Compound Groups, or groups presenting all the cases of the class or classes of defect indicated, whether alone or in combination with other defects. The groups are presented in columns as in Table I.

Compound groups.	Percentages on the number of cases noted with a defect.								Percentages on number seen of all ages.	
	Age 7 years or under.		Age 8—10 last birthday.		Age 11 or over last birthday.		Children of all ages.			
	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
A +. Developmental defect alone or in combination	58.9	66.2	48.9	46.5	48.3	36.4	26.9	23.6	8.7	6.8
B +. Nerve-signs alone or in combination	53.3	45.9	69.2	68.8	69.2	74.8	39.6	38.9	10.8	8.4
C +. Low nutrition alone or in combination	25.5	35.1	15.3	20.4	9.2	15.3	3.8	5.1	2.8	3.2
AB +. Developmental and nerve alone or otherwise	18.7	18.9	21.2	19.2	19.5	16.0	16.6	13.4	3.3	2.5
AC +. Developmental and low nutrition alone or otherwise	13.4	23.2	7.8	10.3	3.6	5.4	5.0	8.5	1.4	1.8
BC +. Nerve and nutrition alone or otherwise	10.5	12.6	7.8	10.4	5.2	7.6	4.6	5.5	1.3	1.4
ABC. Developmental, nerve, and low nutrition	4.8	7.5	3.3	4.1	1.6	2.5	3.3	4.8	.6	.6

TABLE III.—For 50,000 children seen 1892-94. Showing co-relations of the classes of defect, and the association in the form of percentages of groups of cases with other class or classes of defect, whether alone or in combination with other defect.

	Percentages on the number of cases noted with a defect.							
	Age 7 years or under.		Age 8-10 last birthday.		Age 11 or over last birthday.		Children of all ages.	
	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
Developmental defect with nerve-sign . . .	31.7	28.5	43.3	41.3	40.5	44.0	38.4	36.3
Developmental defect with low nutrition . . .	22.7	35.0	16.0	22.1	7.5	15.0	16.2	26.5
Developmental with nerve and low nutrition . . .	8.1	11.3	6.9	8.9	3.4	7.0	6.4	9.6
Nerve-signs with developmental defect . . .	35.5	41.2	30.6	27.9	28.2	21.4	31.0	29.1
Nerve-signs with low nutrition . . .	19.6	27.4	11.3	15.1	7.5	10.2	12.3	16.6
Nerve with developmental and low nutrition . . .	9.0	16.4	4.8	6.0	2.4	3.4	5.2	7.7
Low nutrition with developmental defect . . .	52.5	66.1	51.1	50.3	39.3	35.5	49.9	55.6
Low nutrition with nerve-signs . . .	41.1	36.0	51.1	51.1	56.4	49.9	47.1	43.5
Low nutrition with developmental and nerve . . .	18.8	21.4	22.0	20.3	17.9	16.6	19.9	20.3
Developmental and nerve with low nutrition . . .	25.7	39.7	15.9	21.6	8.4	15.9	16.8	26.6
Developmental and nutrition with nerve . . .	35.9	32.4	43.1	40.4	45.6	46.8	39.8	36.4
Nerve-sign and nutrition with developmental defect . . .	45.9	59.5	43.1	39.8	31.8	33.3	42.2	46.6

TABLE IV.—For 50,000 children seen 1892-94. Showing the Primary Groups, or groups presenting only the class or classes of defect indicated. They are presented as components of the compound groups which contain all the cases with the class of defect named. Cases are arranged in columns as age groups, and in the last column for children of all ages. The number of cases is given; also the distribution of the primary groups as percentages on the number in the compound group. Mental dulness as a character in the cases is here omitted.

	Age 7 years or under.				Age 8-10 last birthday.				Age 11 years or over last birthday.				Children of all ages.			
	Number of cases.		Per-centage.		Number of cases.		Per-centage.		Number of cases.		Per-centage.		Number of cases.		Per-centage.	
	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
A. Developmental defect only	447	340	53.7	47.8	412	269	47.5	45.4	337	150	55.3	47.9	1196	759	51.8	46.9
AB. Developmental defect with nerve-signs only	196	123	23.6	17.2	316	192	36.5	32.4	226	116	37.1	37.1	738	431	32.0	26.6
AC. Developmental defect with low nutrition only	121	169	14.5	23.7	79	78	9.1	13.2	25	25	4.1	8.0	225	272	9.7	16.8
ABC. Developmental with nerve-signs and low nutrition	68	81	8.2	11.3	60	53	6.9	9.0	21	22	3.5	7.0	149	156	6.5	9.7
A +. All cases with developmental defect	832	713	100.0	100.0	867	592	100.0	100.0	609	313	100.0	100.0	2308	1618	100.0	100.0
B. Abnormal nerve-signs only	408	236	54.3	47.7	773	551	62.9	62.9	531	462	66.6	71.8	1762	1249	61.8	62.0
AB. Developmental defect with nerve-signs only	196	123	26.1	24.8	316	192	25.8	21.9	226	116	25.9	18.0	738	431	25.9	21.4
BC. Nerve-signs with low nutrition only	80	55	10.6	11.1	79	80	6.4	9.1	45	44	5.1	6.8	204	179	7.1	8.9
ABC. Developmental with nerve-signs and low nutrition	68	81	9.0	16.4	60	53	4.9	0.1	21	22	2.4	3.4	140	156	5.2	7.7

BC. Nerve-signs with low nutrition only	121	106	33.0	42.7	79	79	80	29.0	30.8	45	22	21.1	20.0	212	212	212	212
ABC. Developmental with nerve-signs and low nutrition	80	55	22.2	14.5	79	80	29.0	30.8	45	44	38.5	33.4	204	179	27.3	23.3	
C +. All cases with low nutrition	68	81	18.9	21.5	60	53	22.1	20.4	21	22	17.9	16.6	149	156	19.9	20.2	
AB. Developmental defect with nerve-signs only	360	378	100.0	100.0	272	260	100.0	100.0	117	132	100.0	100.0	749	770	100.0	100.0	
ABC. Developmental with nerve-signs and low nutrition	196	123	74.2	60.3	316	192	84.0	78.3	226	116	91.5	84.1	738	431	83.2	73.4	
AB +. All cases with developmental and nerve-signs	68	81	25.8	39.7	60	53	16.0	21.7	21	22	8.5	15.9	149	156	16.8	26.6	
AC. Developmental defect with low nutrition only	264	204	100.0	100.0	376	245	100.0	100.0	247	138	100.0	100.0	887	587	100.0	100.0	
ABC. Developmental with nerve-signs and low nutrition	121	169	64.0	67.6	79	78	56.8	59.5	25	25	54.3	53.2	225	272	60.1	63.5	
AC +. All cases with developmental and low nutrition	68	81	36.0	32.4	60	53	43.2	40.5	21	22	45.7	46.8	149	156	39.9	36.5	
BC. Nerve-signs with low nutrition only	189	250	100.0	100.0	139	131	100.0	100.0	46	47	100.0	100.0	374	428	100.0	100.0	
ABC. Developmental with nerve-signs and low nutrition	80	55	54.1	40.4	79	80	56.8	60.2	45	44	68.2	66.7	204	179	57.8	53.4	
BC +. All cases with nerve-signs and low nutrition	68	81	45.9	59.6	60	53	43.2	39.8	21	22	31.8	33.3	149	156	42.2	46.6	
	148	136	100.0	100.0	139	133	100.0	100.0	66	66	100.0	100.0	353	335	100.0	100.0	

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 133.)

CASE OF SUBCLAVIAN ANEURISM

SUCCESSFULLY TREATED BY LIGATURE

BY

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THIS was a case of an aneurism of the third part of the right subclavian, for which the first part of the artery was ligatured twice, and on the second occasion was followed by a distal ligature to the first part of the axillary. A spare healthy man, aged 54, was sent to me by Dr. Sharkey in February, 1896, with the statement that so far as could be ascertained, by physical examination of his chest, the heart and aorta were free from disease.

He had an aneurism of the right subclavian in its third part, but no other evidence of arterial disease. His urine was normal, and there was no history of syphilis. His occupation was that of a cooper, which had necessitated an excessive use of the right arm and shoulder for the saw and hammer.

Three years ago after a long walk he had pain in the right shoulder, which he attributed to rheumatism. After a short rest the pain disappeared, but after any

violent exercise it returned, and the intervals between the recurrences of pain became shorter. The pain, too, passed down the arm until it reached the tips of his fingers. He then tried rubbing his arm with oils, and used an electric battery ; but finding the pain became worse he consulted a surgeon, who told him that he had an aneurism. This was in May, 1895.

On examination in February, 1896, a small sacculated aneurism was found in the course of the third part of the right subclavian artery, reaching from the outer border of the scalenus anticus to the clavicle. It was about the size of a large walnut. The pulsation of the right radial artery was occasionally smaller than the left, but not persistently so. He had no pain in the arm or hand so long as he was kept at rest.

As far as one could tell before the operation the aneurism did not extend beneath the scalenus anticus ; and on the inner side of this muscle there was no difference between the two sides of the neck. It therefore seemed to be a very suitable case for the ligature of the second part of the subclavian, or at the junction of the first and second part.

On March 18th, 1896, an operation was undertaken for ligature of the subclavian, on the proximal side of the aneurism. The skin being drawn downwards, an incision was made from the sterno-clavicular articulation to the outer third of the clavicle. When the skin wound had been retracted upwards, the clavicular part of the sterno-mastoid muscle was completely divided. The internal jugular vein then came into view, and was retracted inwards. There was some little difficulty in defining the fibres of the scalenus anticus, as they appeared to be blended with the thickened fascia on the outer surface of the aneurism.

I felt it wiser, therefore, to do as Mr. Godlee had done in a similar case ('*Med.-Chir. Trans.*' vol. lxxv, p. 275) namely, divide the scalenus anticus a little higher in the neck, above the aneurism, where the muscle could be

distinctly defined from the external coat of the aneurism.

The phrenic nerve was easily recognised and drawn inwards with the internal jugular vein. When the scalenus anticus had been completely divided, it was seen that the aneurism really involved a portion of the second part of the subclavian, and that the ligature must be applied at the junction of the first and second part of this vessel. One branch was given off at this spot, and was directed upwards and backwards towards the neck. It was probably the deep cervical judging from its direction, and arose from the superior intercostal.

This artery was first ligatured; and then the subclavian on its proximal side, exactly at the inner margin of the scalenus anticus. Neither the transverse cervical nor the supra-scapular were seen; the former probably arose from that portion of the subclavian which was involved in the aneurism.

At the seat of ligature the artery appeared healthy.

The material used for the ligatures was carefully prepared goldbeater's skin, which had been kindly provided by Messrs. Ballance and Edmunds.

The aneurism needle was in each case first passed beneath the vessel and then threaded. A long ligature was drawn through the eye of the needle to its centre. The needle being withdrawn from beneath the vessel the loop was divided; two ligatures were thus left beneath the artery. These were then tied in the manner described by Messrs. Ballance and Edmunds as the "stay knot." Sufficient force was used to completely stop the pulsation in the parts beyond, but no attempt was made to divide the coats.

The wound was closed with horsehair sutures without drainage, and without the employment of antiseptics; the surface was covered with sterilised gauze.

The first dressing was changed on the 23rd March—five days after operation,—as the temperature was not quite normal. Some sutures were removed, as there

FIG. 1.

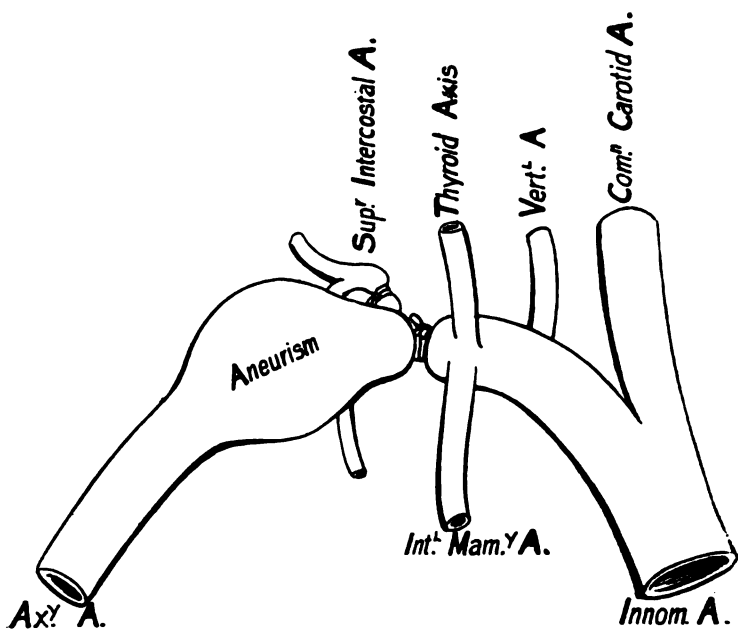
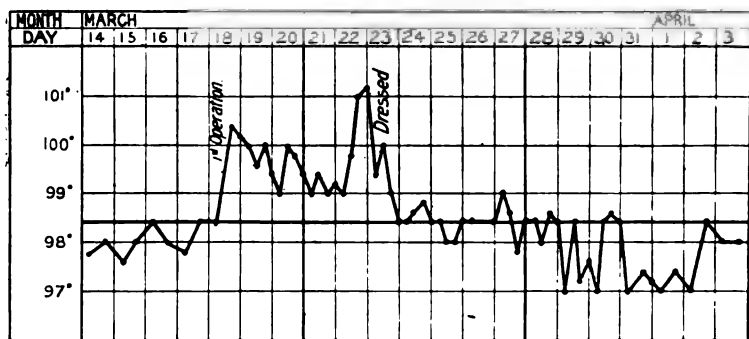


Diagram to show the seat of ligature in the first operation. Goldbeater's skin ligature.



Temperature chart after first operation.

was a little blood and pus beneath the incision; the suppuration, however, appeared to be quite superficial.

The wound was dressed again on the 25th, when the temperature was normal. Pulsation was now for the first time felt in the aneurism, but disappeared again a few days afterwards.

The suppuration at the surface of the wound continued for about ten days, but was quite insignificant in the amount of pus produced, and in its extent.

On April 7th there was still no pulsation in the aneurismal sac, but a *bruit* was detected.

On the 21st, after careful examination, pulsation in the aneurism was distinctly made out, but the radial artery could not be felt.

On the 28th, about six weeks after operation, both the aneurism and the radial artery could be felt pulsating as they did before the operation.

It was now clear that our treatment had failed, either from absorption of the animal ligature or from its being tied with insufficient tightness, or possibly from a too brisk collateral circulation.

Digital compression of the highest part of the axillary artery produced no diminution in the force of the pulsation in the aneurism. This compression would probably exclude all the axillary anastomosis. I was forced, therefore, to conclude that either the ligature had been absorbed or that it had been insufficiently tied. Against the latter alternative was the fact that pulsation had been arrested at the time of the operation and was not felt till seven days afterwards, and that the pulsation again disappeared for two or three weeks, and was not distinct till five or six weeks after operation. On the whole, therefore, I thought it most likely that the ligature had been absorbed.

Sepsis had, it is true, taken place at the surface of the wound, but so far as one could tell, the suppuration had not extended to any depth, and was soon over. If it had reached so far as the seat of ligature, it would

account for premature softening of the ligature. But the artery was not pervious for five weeks, and another week elapsed before the pulsation was fully re-established. This is just about the time at which the animal ligature should disappear, and be replaced by young connective tissue. Messrs. Ballance and Edmunds have shown by their experimental work this to be about the time in an aseptic wound for the animal ligature to be replaced, and that if suppuration occurs the absorption of the ligature takes place still earlier. This raises the important question whether the animal ligature is trustworthy for large arteries where the blood pressure is considerable. For if sepsis was not accountable for the absorption of the ligature, it is clear that an animal material is not satisfactory for the ligature of a large artery in continuity—unless, indeed, there are still graver objections to the use of a silk ligature.

As the aneurism was now the same size as before the operation, I determined to re-ligature the subclavian artery on the proximal side with silk. But I thought it best to wait a few weeks, if it were possible to do so. The man was therefore kept in the recumbent position, and a careful watch kept on the aneurism.

On June 8th, about ten days after the aneurism was recorded as pulsating as forcibly as before, it was noticed that it had perceptibly increased in size, and the man complained of pain down his arm. I decided, therefore, not to wait any longer.

On June 10th the second operation was undertaken. A curved incision was made above the scar of former operation, terminating just below the middle of the episternal notch. The clavicular part of the sterno-mastoid muscle was again divided, but at a higher level. The sternal origin of this muscle having been retracted inwards, the sterno-hyoid and thyroid muscles were exposed and divided.

The internal jugular vein and carotid artery were then seen lying one on each side of the centre of the wound,

FIG. 2.

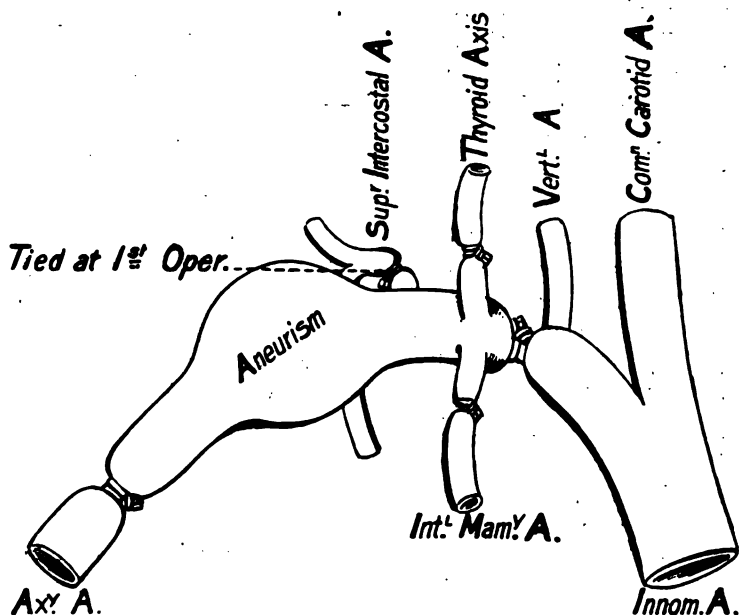
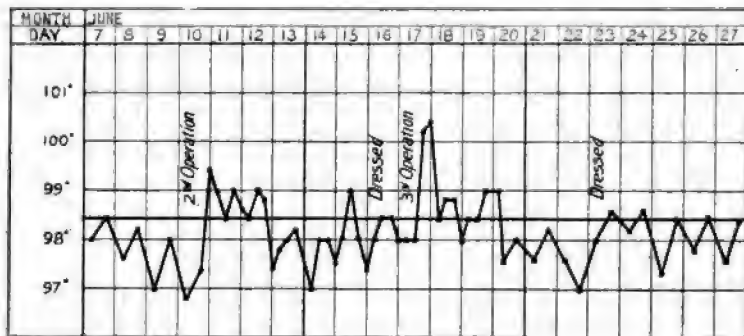


Diagram to show the ligatures applied in the second and third operations. Floss silk ligatures. The supra-scapular and transversalis colli are not shown, as they were not seen. It is impossible to say positively from which vessels they arose.



Temperature chart after second and third operations.

which crossed them obliquely. The interval between these two structures was carefully enlarged, till the pneumogastric nerve was recognised.

When the carotid artery with the pneumogastric had been drawn inwards with a retractor, and the internal jugular vein with the phrenic—which was not, however, seen—drawn outwards in a similar manner, there was a very good space for the further steps of the operation.

The first part of the subclavian artery could now be felt and traced onwards to the aneurism.

The spot where the artery had been previously ligatured was easily recognised, and was normal in size. It was surrounded by a small amount of connective tissue. To separate this tissue for the purpose of passing a needle might, I thought, lead to a wound of the pleura; and on the proximal side the subclavian, with several of its branches, could be seen lying quite loose in its cellular bed. The vertebral vein was now divided, as it was in the way.

The branches of the subclavian artery, which could be seen, were the internal mammary and the thyroid axis.

The subclavian artery was first of all tied with a double floss silk ligature on the proximal side of these two branches, in the same manner as in the first operation. The thyroid axis and the internal mammary were then similarly tied with the same material.

The pulsation in the aneurism, and in the radial artery, ceased as the ligature on the main vessel was being tightened, but no extra effort was made to rupture the coats.

The wound was closed without drainage, and without the use of any antiseptic solution, and dressed with cyanide gauze. The operation, which had occupied an hour, would have been a long and tedious business if there had been much inflammatory tissue as a result of the first operation. But, as has been shown, there was really very little—a fact which goes far to support the contention that the organisms present at the surface of

the wound, after the first operation, had not penetrated to the deeper parts.

After the second operation the temperature remained normal, but the dressing was removed on the 16th to see if the aneurism remained pulseless.

I had almost made up my mind at the second operation to tie the first part of the axillary as well as the subclavian, so as to cut off the collateral supply from the intercostal arteries, which might have been formed after the first operation. But on the whole I thought it better to wait a week to see the result of tying the subclavian with silk.

When, therefore, the dressing was removed I was not surprised to find the aneurism with distinct pulsation and bruit. The wound had healed, and the sutures were removed.

On the following day, June 17th, the third operation was undertaken, namely, the ligature of the first part of the axillary artery, so as to cut off the collateral supply from the distal side of the aneurism. An incision was made immediately below the clavicle, dividing the clavicular fibres of the pectoralis major. The artery was reached above the pectoralis minor, and tied as close under the clavicle as possible.

A double floss silk ligature was passed and divided. Then each was tied separately so as to avoid so large a knot as was made in the previous ligatures, but in tightening the first loops of the two knots the ends on each side were pulled simultaneously as in the "stay-knot."

The pulsation in the sac was materially diminished, but not entirely obliterated. Thinking there might be an enlarged abnormal branch between the aneurism and the scapula, such as the posterior scapular or supra-scapular artery, I made an incision in this position. The supra-scapular nerve was seen, but no accompanying artery. A finger was also introduced which would have felt any enlarged vessel, but nothing of the kind was found.

The wounds were closed without drainage or antiseptics, and dressed with cyanide gauze.

The temperature after this operation remained practically normal, but the dressing was changed on the 22nd to examine the aneurism. The wound was healed, and I thought there was no pulsation in the sac, but the next day, on re-examination, a very faint pulsation could be felt.

The subsequent history can be shortly told. The wounds healed by first intention; the aneurism began to diminish in size, and soon ceased to give even the faintest pulsation.

On July 13th I noted the fact that no pulsation had been felt for some days, and the aneurism was shrinking in size.

On August 3rd there was no swelling to be felt above the clavicle, so that the aneurism, by that date, may be said to have ceased to exist.

The patient went home on August 8th. Dr. Sharkey examined him before he left, and found no alteration in the physical signs in the chest. The pulse at the wrist had not returned, but there was no swelling in the arm or hand, and there was no pain or tenderness.

He was again seen in October, when the same condition existed, namely, complete absence of pulsation above the clavicle and no radial pulse at the wrist.

Remarks.—I do not wish to draw any definite conclusions from this interesting case, as it appears to me that there are not enough records as yet of operations upon subclavian aneurisms, which have been conducted on modern lines of treatment, to enable one to state, with any degree of accuracy, the particular method which is most likely to be successful.

I would like, therefore, to leave this case with as few observations as possible upon the details of treatment which I felt called upon to carry out during the progress of events which have been described.

For anyone who is interested in the history of the

operative treatment of aneurisms of the third portion of the subclavian artery there is a very interesting article by Dr. Edmund Souchon, in the 'Annals of Surgery for 1895,' vol. ii, p. 545 and 743. Here also the treatment in the future is foreshadowed.

With regard to my own case I would summarise the details of the treatment in this way.

(1) There was sufficient room to apply a ligature on the proximal side of the aneurism, provided the coats of the artery were not divided by a tight ligature. If the latter method of applying a ligature were thought to be best, then the artery should be, in my opinion, divided between the two ligatures. The position of the artery to be tied in this case—namely, the root of the neck—makes this an undesirable method. The artery was not therefore tied with sufficient tightness to divide the coats in any of the operations recorded in this paper.

(2) An animal ligature appeared to me to be the best material, but the failure in this case of procuring occlusion of the vessel before the ligature disappeared, suggests that a more permanent material at the root of the neck would have been safer.

Some surgeons may say that if, with the animal ligature, the coats had been divided, such failure of procuring occlusion would not have occurred. But the large number of vessels arising in the immediate neighbourhood of the ligature, and the high blood pressure at this spot, induce me to think that an aneurism at the seat of ligature would not be an improbable accident as the result of the injured coats.

(3) It is probable that if the first ligature had been of silk instead of an animal material, the subsequent operations would have been unnecessary.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 141.)



CASE OF PHARYNGEAL ABSCESS

HÆMORRHAGE; LIGATURE OF CAROTID ARTERIES

BY

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THIS was a case of very severe hæmorrhage from an abscess in the roof of the pharynx above the right tonsil, which was eventually successfully arrested by the ligature of the common, external, and internal carotid arteries.

A man aged 28, a victualler by trade, was admitted into St. Thomas's Hospital, under Dr. Sharkey, on June 20th, 1896. He first felt pain in his limbs and sore throat on the 18th, two days before applying for admission.

His temperature on admission was 101° F. His symptoms rather suggested the onset of rheumatic fever as the cause of his illness, for, on examination, there was very little swelling to be seen in the throat. On June 21st, the day after admission, he bled rather profusely from the mouth, and it was thought, as there was a swelling above the right tonsil, that an abscess in this position was the source of the hæmorrhage. Moreover, the blood was seen to come from the right side.

He bled again on the 22nd, and on the 23rd Dr. Sharkey sent for me to see the case. His temperature was still high.

On examination I found that there was a swelling above and behind the right tonsil, but that the tonsil itself was not sufficiently large or vascular to be the seat of an abscess. There was no swelling externally in the neck, but some tenderness on pressure at the right angle of the jaw, and above this towards the base of the skull. The patient was a short-necked plethoric man, and judging from his appearance, addicted to a large consumption of beer. I made a digital examination of the posterior nares, and found a round smooth swelling above the right tonsil, filling up the space round the opening of the right Eustachian tube, and almost blocking the right posterior naris. In the middle of this swelling I thought I could feel a small opening. There was no bleeding at the time of my examination, but the same evening the hæmorrhage was repeated.

The next day, June 24th, he was taken to the operating theatre for a more complete examination. After the administration of an anæsthetic the soft palate was divided in the median line, and the two halves held apart by fine silk sutures. With the patient's head hanging over the edge of the table, a small round opening could be seen in the centre of the swelling, which was situated quite in the roof of the pharynx on the right side. The opening was between the margin of the right posterior naris and the Eustachian tube, but was too small to admit the tip of my finger. It was therefore enlarged backwards with a blunt-pointed bistoury. A finger was introduced into this wound, and a small abscess cavity found, extending deeply towards the neck, below the right Eustachian tube. Up to this point there had been no bleeding, but as the finger was withdrawn some oozing took place, which made me think that the hæmorrhage might have come from the jugular vein. The cavity was therefore plugged with cyanide gauze, and the patient sent

back to bed, with the palate widely open for any future emergency.

The same night he bled furiously, and when Mr. Abbott, the resident assistant surgeon, arrived, he found that two porringers, which are each capable of holding a pint, were full of bright red blood. Mr. Abbott quickly removed the plug, and introduced another more firmly. The man was extremely faint, but the hæmorrhage had been successfully arrested for the time.

On my visit next day, the 25th, I found the man so blanched and anæmic that I felt sure he would not stand another attack of hæmorrhage, and that it would not be wise to let him run the risk. I was also pretty confident, from the character of the last attack of hæmorrhage during the preceding night, that the bleeding was from the internal carotid artery.

The cavity which I had felt was too high for the suppuration to have involved the tonsillar branch of the facial artery, and was, I thought, too severe for the ascending pharyngeal. Still it was a possibility, but the operation I proposed to do would secure this artery as well as the internal carotid.

The man was therefore removed to the theatre, and the operation for ligature at the bifurcation of the common carotid undertaken, with the assistance of Mr. Abbott and Messrs. Crouch and Dyball, the two house surgeons.

Having exposed the carotid at its bifurcation the internal carotid was first ligatured, then the external, and finally the common carotid, all the ligatures being applied as close to the bifurcation as possible. Goldbeaters' skin was used for this purpose, a double ligature being passed in each case and tied as a stay-knot. The coats were not ruptured as far as one can tell, but the operation was necessarily a hurried one.

If the common carotid alone had been tied, the external carotid might have quickly brought blood round past the bifurcation to the internal carotid. This probability has

been well established by Mr. Pitts in 'St. Thomas's Hospital Reports,' vol. xii, p. 131.

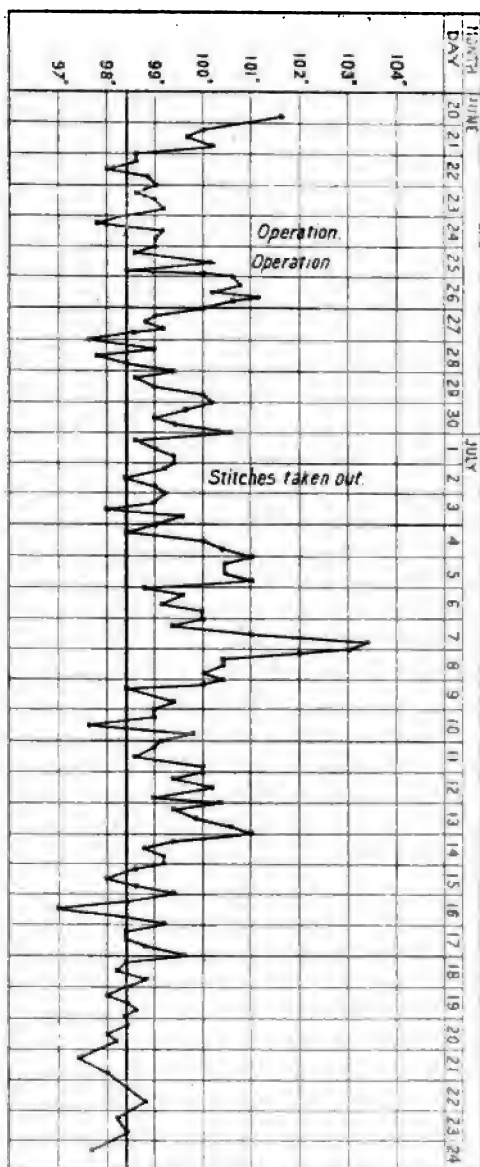
It was therefore necessary to tie one or other of these arteries as well as the common carotid; and to tie both of them, made the obliteration of the channel more secure. It was also necessary to tie the common carotid to ensure the closure of the ascending pharyngeal artery. I had therefore made up my mind to ligature all three arteries at the bifurcation if the operation became necessary.

The man ceased breathing at the commencement of the operation, but recovered quickly with artificial respiration. The whole operation had to be very rapidly performed, as the patient was in a most feeble condition. Two pints of a saline solution were thrown into the most prominent vein at the bend of the elbow by Mr. Abbott whilst I was suturing the wound, and applying a fresh plug to the abscess in the pharynx.

There is nothing to record in the after history of the case. The wound in the neck healed by first intention; the hole in the pharynx gradually closed, and the man had no further hæmorrhage. On July 16th examination with the finger proved that the pharynx had healed; the divided palate had also granulated up so far that it seemed probable an operation for its closure would be unnecessary. On July 23rd he was perfectly well, but still very anæmic, and was therefore sent on July 25th to a convalescent home.

When called upon to treat a serious case of hæmorrhage from the pharynx it seems impossible to determine upon the exact vessel which is involved. It may be either the tonsillar branch of the facial, the ascending pharyngeal, or the internal carotid itself, which is the seat of the hæmorrhage.

Under these circumstances I would submit that it is the safest practice, if an operation has to be done to save life, to tie all three arteries at once, namely, the common carotid and its two branches, as it does not add to the



risks, but rather diminishes them, to cut off all the collateral supply by one operation, leaving only the anastomosis from the opposite side. If the bifurcation is exposed, the operation is not materially lengthened by the addition of the third ligature.

With regard to the tightness of the ligature and the rupture of the coats, it would seem best always to avoid this injury in large arteries if it be possible; and a permanent obliteration of the vessel is not so essential as it is in the treatment of an aneurism. For the same reason an animal ligature is quite sufficient for the purpose, for even if it were to melt away in five weeks' time, and the channel be restored, the abscess has had sufficient time to heal, and close the opening in the bleeding vessel.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 144.)

ON IRREDUCIBLE HERNIA

BY

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Received February 8th—Read May 26th, 1897

IN this paper I have analysed eighty-five cases of "irreducible hernia" with a view to ascertaining the causes of the irreducibility and the treatment necessary to overcome it; and I have ventured to bring the results before the Royal Medical and Chirurgical Society, because it seems to me that the conclusions to be deduced from them are somewhat different from those usually set forth in the text-books of Surgery. The term "irreducible hernia" is used in the commonly accepted technical sense; that is to say, a hernia of which a part, or the whole, cannot be returned into the abdominal cavity, but which, nevertheless, exhibits none of the symptoms of strangulation or obstruction.

An irreducible hernia may, however, and often does, become strangulated; and this is one of the dangers to which persons the subject of irreducible hernia are liable; but when spoken of as simply "irreducible," it is implied that neither the circulation nor the function of the part protruded is materially interfered with.

The cases I have tabulated include all the cases of irreducible hernia operated upon in St. George's Hospital during the last ten years, together with a few from my private practice.

It is difficult to estimate from hospital practice what proportion the irreducible variety bears to other kinds of hernia, because it is chiefly the severe and intractable cases which seek admission.

M. Paul Berger¹ gives the following table showing the number of cases of irreducible hernia met with among 10,000 herniæ of all kinds :

	Inguinal.		Femoral.		Um-bilical.	Epi-gastric.	Ventral.	Trau-matic.	Total.
	R.	L.	R.	L.					
Men . . .	123	87	20	19	8	17	—	1	275
Women . . .	18	13	64	38	164	2	6	2	307
Total . . .	141	100	84	57	172	19	6	3	582

i. e. nearly 6 per cent. of the whole number were irreducible. Mr. Macready gives the proportion of irreducible cases as about 1 per cent. of inguinal herniæ, and about 10 per cent. of femoral.²

These statistics show that the condition is by no means uncommon, and my own tables prove that it is attended with considerable danger : for of eighty-five cases of irreducible hernia requiring operation for various reasons, strangulation had occurred in twenty-five : and of the whole number of cases of strangulated hernia operated upon in St. George's Hospital during ten years, 12½ per cent. had been for varying periods simply irreducible.

Besides the danger of strangulation, an irreducible hernia is often the cause of much inconvenience and distress. Excluding the cases in which operation was imperative

¹ 'Dix mille observations de Hernies,' par Paul Berger, Paris, 1896, p. 173.

² 'Treatise on Ruptures,' p. 218.

because of strangulation, the remainder sought operation either because their condition interfered with the pursuit of their ordinary avocation, or because of the pain or inconvenience caused by the hernia.

Nearly all herniæ which are irreducible tend to increase in spite of any sort of bandage or support which may be worn; this is, of course, especially the case if the patient suffers from cough, or follows a laborious trade. Thus, in the course of time, many irreducible herniæ attain most inconvenient dimensions; the weight of the tumour, the sense of dragging on the parts above, and the soreness of the skin from friction, causing constant discomfort. Moreover, the larger a hernia, the more liable is it to injury, and the greater is the danger should operation become necessary. Dyspepsia, constipation, and attacks of abdominal pain are other troubles met with in connection with irreducible hernia.

If now we investigate the reason of the irreducibility of the herniæ tabulated, we find that it was due in nearly every case to adhesion or other changes in the protruded viscera, not to any special condition of the containing sac. Reduction of a hernia is, however, occasionally prevented by thickening or contraction of the neck of the sac, and by hour-glass contraction of the body of the sac; but these conditions must be rare, and were met with in only two of the cases I have tabulated. The great size of a hernia of long duration may, no doubt, prevent the possibility of returning the protruded viscera into the abdomen from which they have long been absent; but this, again, is a condition not very often seen in the present day.

The changes observed in the contents of the sac, which rendered the herniæ irreducible, were of various kinds; but by far the most frequent cause of irreducibility was the presence of omentum which had either contracted adhesions to the sac, or had undergone other changes preventing its reduction. Of the eighty-five cases tabulated, sixty-seven contained omentum in the sac; in thirty-four of these the omentum was adherent to the interior of the sac; in the

remaining thirty-three the omentum had become thickened, indurated, and matted together in a manner which rendered its return through the abdominal ring impossible. In one case (No. 74) the omentum, besides having become adherent and nodular, contained several cysts within its folds, a condition effectually preventing reduction.

In one case (No. 77) a lady had had an irreducible hernia a year, which had recently increased in size, and had become painful and tender ; no truss could be worn. She suffered much from constipation and dyspepsia. Operation was advised, and on opening the sac it was found to contain a piece of omentum, which was twisted spirally, so that the distal part of it was much congested and swollen. After untwisting the omentum it was returned, and an operation for the radical cure of the hernia was then performed. In this case it was evident that the rotation of the omentum had taken place recently, the congested folds had not become adherent, and could be again spread out ; but it seems to me probable that this twisting may be not uncommon, and may account for the adherent and indurated condition in which the omentum is often found in cases in which no truss has been worn.

In one case (No. 55), reduction of the hernia was prevented by enormous thickening and induration of the mesentery of a large coil of intestine. The patient, a stout man aged sixty-six, had a large scrotal hernia of three years' duration, which had been irreducible for a year, and was a source of much trouble and pain. He also suffered from severe cough, due to chronic bronchitis. He was kept in bed, of which the lower end was raised, pressure was applied to the scrotum, he was freely purged, and attempts were made to reduce the hernia. This treatment was persisted in for more than a month, as it was felt that the large size of the hernia, and the condition of the patient, rendered the case a somewhat unfavorable one for operation ; but as not the least effect was produced on the hernia an operation was decided upon. The sac contained no omentum, but there was a large coil

of small intestine, the mesentery of which was as thick as the palm of the hand, very hard and quite inflexible. This condition of the mesentery rendered reduction of the bowel impossible, until the ring had been considerably enlarged by incision. The bowel having been then with some difficulty returned, the sac was drawn down, divided and ligatured, the pillars of the ring laced together, and the wound closed with deep and superficial sutures. The man made a good recovery.

In twenty-two cases the hernia consisted of irreducible intestine as well as omentum. In fifteen of these the bowel became strangulated. In three cases both omentum and bowel were adherent to the sac; in two of these strangulation of the bowel occurred. Of the twenty-two cases containing bowel and omentum, in eight the omentum only was adherent to the sac: in eleven the omentum was simply thickened and its folds adherent; in one the intestine was adherent to the sac, and the omentum thickened and adherent, but not to the sac; in only four of the twenty-two was the reduction of the bowel prevented by its adhesion to the sac.

Of the eighty-five cases tabulated, twelve were irreducible because of adhesions of the bowel to the sac, and four because of adhesion of the coils of intestine. That these are both conditions of serious danger is apparent from the fact that of the twelve cases in which the intestine was adherent to the sac, the bowel became strangulated in five, of which two died; and of the four cases in which the coils of intestine were adherent, strangulation of the bowel occurred in three, which were all fatal.

One of these last (No. 78) was that of a man aged fifty-four who had been operated upon for strangulated hernia five years previously, when the coils of bowel in the sac were found firmly adherent and were not returned into the abdomen. The hernia was of twenty-nine years' duration, and had been for many years irreducible. The man had been frequently in danger from strangulation and obstruction, and constantly suffered so much discomfort that he

was anxious for some further attempt to be made for his relief. The sac being opened, the coils of bowel were found to be firmly bound together by old adhesions over a large part of their surface and quite inseparable; there were also firm adhesions to the sac. The sac was dissected from its surroundings and from the bowel, and cut away, portions being left attached to the bowel. The intestine was returned with considerable difficulty, the neck of the sac ligatured, and the pillars of the ring laced together.

The patient died on the fifth day from paralysis of the intestine. The case well illustrates the dangers of leaving a hernia unreduced. It is impossible to say what was the cause of irreducibility when the hernia first occurred, but it could probably have been overcome by an operation of very little risk; whereas the subsequent adhesions of the intestine rendered reduction both difficult, dangerous, and of doubtful benefit.

I am inclined to think that in this case it would have been better to have resected the adherent coil; for the adhesions were so firm and the flexion so acute as to constitute an almost inevitable danger in the future.

In only two cases (Nos. 60 and 82) did the obstacle to reduction appear to have been simply the large amount of protruded bowel; and one of these (No. 60) proved fatal from paralysis of the bowel, probably due to the unavoidably prolonged handling necessitated by the difficulty in returning the intestine into the abdomen.

In the second case (No. 82) the bowel became strangulated, was operated upon, and the patient recovered.

In one other case (No. 80) the irreducibility depended partly upon the presence of thickened and adherent omentum, and partly upon the large amount of intestine in the sac. Here again the bowel became strangulated, the patient was operated upon and recovered. But all three cases show the dangers belonging to large irreducible herniæ, their liability to strangulation and the greater risk of operation, in consequence of the prolonged

handling of the bowel frequently rendered necessary by the difficulty of reduction.

Of the eighty-five cases, in only three was reduction prevented by the condition of the sac.

In one (No. 6) a band stretched across the sac, and perhaps this would have prevented reduction of the bowel ; but there was also adhesion of the coils of intestine, such as to render its return impossible without operation. Strangulation of the bowel occurred and proved fatal.

In a second case (No. 39) a constriction of the sac appeared to have been the only obstacle to reduction. After several years of irreducibility, strangulation of the bowel occurred, the patient was operated upon, and recovered.

In the third case (No. 56) the sac was thickened by layers of laminated clot like those in an aneurism, and this was the only discoverable obstacle to reduction of the bowel. The sac was removed, and an operation for the radical cure of the hernia performed. The patient had worn a variety of trusses which were ineffectual and uncomfortable, and the hernia had lately become painful and tender. I presume, therefore, that the blood-clot was due to hæmorrhage caused by the pressure of the truss on unreduced bowel.

One other condition remains to be mentioned,—a case of ventral hernia of subperitoneal fat (No. 73). A man aged forty-two years had a tumour the size of a hen's egg between the ensiform cartilage and the umbilicus. It had a distinct impulse on coughing, was irreducible, and was dull to percussion. He complained of pain in the tumour on stooping. The tumour was exposed by incision, and found to be fat protruding through an opening between the recti muscles. The tumour was removed, the edges of the opening freshened and laced together, and the wound closed by a continuous suture.

It will be seen from an analysis of the preceding eighty-five cases of irreducible hernia that the causes of the irreducibility may be arranged as follows :

Table showing Cause of Irreducibility in Eighty-five Cases of Irreducible Hernia.¹

	Cases.
Thickened and adherent omentum . . .	31
Adhesion of omentum to sac . . .	34
Twisting of omentum . . .	1
Cysts in omentum . . .	1
<hr/>	
Total number of cases in which irreducibility due to condition of omentum . . .	67
<hr/>	
Adhesion of intestine to sac . . .	12
Adhesion of coils of intestine . . .	4
Large amount of intestine . . .	3
<hr/>	
Total number of cases due to conditions affecting intestine . . .	19
<hr/>	
Due to thickening of mesentery . . .	1
Due to conditions affecting the sac only . . .	3
Ventral hernia of subperitoneal fat . . .	1

From this table it is evident that in the great majority of cases of irreducible hernia, the irreducible material is a piece of omentum which is either adherent to the sac, or has undergone inherent changes which prevent its reduction. When the sac contains both bowel and omentum, the bowel is very often reducible, while the omentum is not, for the bowel does not so easily contract adhesions as does the omentum; but in such cases the condition of the patient is extremely insecure. For adhesion and induration of the omentum and its attachment to the interior of the sac commonly result from attempts to wear a truss upon unreduced omentum; a truss so worn is usually a source of pain, is never efficient, and is frequently discarded; moreover, the omentum forms a kind of guide along which the bowel is apt to descend, and thus at any

¹ In a few cases more than one of the above causes of irreducibility were present.

moment strangulation may occur. Of this among the eighty-five cases there are fourteen examples, with three deaths.

It seems to me, therefore, that in all cases in which after a reasonable attempt at reduction there remains in the sac of a hernia a portion of omentum which cannot be returned, an operation is demanded. For if the omentum is adherent to the sac it is obviously irreducible except by operation, and if it is not adherent it will soon become so if subjected to the pressure of a truss. A truss so worn is inefficient, and the security of the patient is very little greater than if no truss is worn, when there is the constant danger of protrusion and strangulation of the bowel. Another source of danger in cases of irreducible omental hernia is that of internal strangulation of the intestine by a band connected with the omentum in the hernial sac. An example of this is Case 83. A woman was admitted into the hospital with symptoms of acute intestinal obstruction. She had an irreducible hernia consisting only of a piece of omentum adherent to the sac; from this a band extended across a portion of intestine within the abdomen, and thus caused strangulation of the gut. The patient was enormously fat (the subcutaneous fat on the abdominal surface was four inches deep), and she was in the fifth month of pregnancy. Having ascertained that there was no intestine in the sac, the abdomen was opened, the band traced and divided, and the strangulated bowel released. Unfortunately abortion occurred, and she died on the third day. I may mention in connection with this case that the operation was performed without anæsthesia, because of the extreme abdominal distension and the unfavorable condition of the patient. A 1 per cent. solution of cocaine was injected in the line of the skin incision, and the patient stated that she felt no material pain.

Considering, then, the dangers that arise in connection with the presence in a hernial sac of irreducible omentum, and that of the sixty-seven such cases tabulated there was not one in which the omentum could have been reduced

without operation, I would suggest that the proper course in cases of irreducible omental hernia is to cut down upon and open the sac, ligature and remove the adherent or thickened omentum, return the stump into the abdomen, and then to perform an operation for the radical cure.

That the risk of such an operation is very small is proved by the fact that of forty-one cases operated upon in which the sac contained irreducible omentum only, there were but two deaths, one of which (No. 84) was from cerebral hæmorrhage in an alcoholic man three days after the operation; the other (No. 67) was a case of old umbilical hernia in a woman with fatty heart, and in whom there was old thickening and great distension of the bowel, so that presumably the hernia had at some time contained bowel as well as omentum.

If there be intestine in the sac as well as omentum, an attempt should be made to reduce the bowel before operation, and then the omentum should be treated as I have suggested above; but in no case should a truss be applied upon unreduced omentum. To wear a truss upon irreducible intestine is a still more dangerous practice. For the pressure and irritation of the truss are nearly certain to produce inflammatory adhesions between the adjacent coils of intestine, or between the intestine and the interior of the sac, a condition favouring strangulation and otherwise interfering with the function of the bowel, and adding greatly to the dangers of any operation that may subsequently become necessary. This is shown in a striking manner by the fact that of the sixteen cases in which the hernia was irreducible because of adhesions of the bowel, strangulation of the bowel occurred in eight, and of these five were fatal.

I maintain, therefore, that cases of intestinal hernia which cannot be completely reduced so that a truss can be efficiently applied, should be looked upon as of sufficient gravity to necessitate the patient being confined to bed and subjected to surgical treatment, so that every chance may be given of replacing the bowel within the

abdominal cavity; and that in no such case should the patient be allowed to go about, or wear a truss. This should be quite irrespective of whether the hernia gives rise to any functional disturbance. For it matters not how long a hernia has been simply irreducible, it may at any moment become strangulated and the patient's life endangered. This happened in twenty-five of the eighty-five cases; and in one of these (No. 47) the patient, a woman seventy-eight years of age, had had an irreducible femoral hernia for forty years without it troubling her much, until at last it became strangulated so tightly that a few more hours' constriction would have produced gangrene, and when I operated I was in some doubt as to whether it would be safe to return the gut.

In connection with the change from the irreducible to the strangulated condition I would venture to allude to the diagnostic importance of the occurrence of vomiting. This is not a symptom belonging to irreducible hernia, though constipation and flatulent distension are common, so that I would strongly insist that a person the subject of irreducible hernia (of however long duration) if attacked with persistent vomiting should be treated without delay as a case of strangulated hernia.

A hernia, however, that is simply irreducible, and which is unaccompanied by any untoward symptoms, may be treated for a time in the hope that it may become reducible. And for this, I think, generally the best plan is to keep the patient in bed, recumbent upon a mattress sloping towards the shoulders, while gentle pressure is made upon the hernia by an elastic bandage over a layer of wool. A fluid and spare diet should be given, and the bowels kept freely purged. The cases in which this is most likely to succeed are those in which there is only bowel in the sac and in which no truss has been worn. But if after three or four weeks of such treatment any part of the hernia remains irreducible, I have no doubt that an operation should be performed; for the irreducibility will probably be found to depend upon adhesions or

some condition not remediable except by operation, and the dangers of an irreducible hernia are much greater than those which belong to the operation for its relief. Moreover, an operation for the radical cure of the hernia can often be undertaken at the same time.

The only exceptions to this rule should, I think, be very large old herniæ, in which a great quantity of the intestine has been so long outside the abdominal cavity that it might be difficult or impossible to make room for it within. Moreover, in very large herniæ the amount of handling necessary for returning the bowel makes the operation a more serious one (as in Case 60). This is an additional argument for operating upon irreducible herniæ before they attain a large size, and also for endeavouring to return as much as may be possible of all large herniæ before operating upon the irreducible remainder. It will be found that there are very few herniæ of the bowel only, even of large size, which are not reducible by the measures I have indicated *as long as the bowel has formed no adhesions*, and this will depend chiefly upon whether a truss has been worn over unreduced bowel. And even when adhesions have formed it is often possible to return a considerable part of a large hernia.

An example of the size which a neglected hernia may attain, and showing also how large a hernia may yet be reducible, is represented in the accompanying illustration.¹ The patient, a woman aged forty-three years, was admitted under my care into St. George's Hospital, in 1895. She had an enormous inguinal hernia reaching halfway down the thigh, and across the middle line. The size and weight of the hernia gave her much distress, which was aggravated by the soreness of the skin produced by friction of the tumour against the thighs. The hernia had existed eight years, and had been irreducible for more than a year. She had worn no truss, and the hernia steadily increased in size. During the last year she had had frequent attacks

¹ I am indebted to the kindness of my colleague Mr. Clinton Dent for the photograph of this remarkable hernia.

of pain, constipation, and occasional sickness. The tumour was resonant and tense, and there was impulse conveyed to it on coughing, but attempts at reduction made at first no impression upon it. She was kept recumbent with the foot of the bed raised, given a spare diet, and purged.

Ice, and subsequently gentle pressure, were applied to the tumour. In three weeks the bowel had all been returned into the abdomen, but there remained a very large pendulous mass of thickened and ulcerated skin which had been stretched over the hernia. The sac was opened and ascertained to be empty, its neck ligatured, and the whole of the pendulous skin together with the sac cut away. The pillars of the ring were laced together, and the wound closed. The woman made an excellent recovery, and there was no protrusion at the inguinal ring, but as her abdominal muscles were very lax, I thought it wise to apply a truss.

It will be observed that in this case the hernia consisted entirely of bowel, and that no truss had been worn, and therefore it was that in spite of its large size and its having been long irreducible, I had hope of it being eventually returnable, as proved to be the case.

There remain for consideration the three cases in which the irreducibility depended upon conditions affecting the sac only. In one case there was "hour-glass" contraction, in one a band stretched across the sac, and in one the sac was thickened by laminated clot. These conditions I have shown to be of rare occurrence, and it may suffice to point out that each of them was such as could be remediable only by operation.

The foregoing facts, and the arguments based thereupon will, I think, justify the following conclusions :

1. That irreducible hernia is a condition of serious danger, the gravity of which increases with its duration or neglect.

2. That the application of a truss upon an irreducible hernia is not only useless but harmful.

3. That irreducible herniæ which consist wholly or in part of bowel are very apt to become strangulated.

4. That herniæ consisting wholly of bowel, upon which no truss has been worn, may (even when of large size and of considerable duration) generally be returned by appropriate treatment.

5. That of the cases of hernia in which proper treatment fails to obtain reduction, the great majority are irreducible because of adhesions of, or changes in, the protruded viscera, conditions which are remediable only by operation.

6. That the most common cause of irreducibility is the presence in the sac of adherent omentum.

7. That the presence of irreducible omentum in the sac of a hernia is a source of constant danger, which can nevertheless be remedied by an operation of extremely small risk.

8. That herniæ containing irreducible bowel are more dangerous than those containing only omentum ; and that as the danger of operation increases with the magnitude of the hernia, and with the occurrence of adhesions, these herniæ should be operated upon as soon as they are proved by the failure of proper treatment to be irreducible.

Analysis of Eighty-five Cases of Irreducible Hernia.

No.	Sex and age.	Situation of hernia.	Duration		Contents of sac	Cause of irreducibility.	Treatment.	Result.
			of hernia.	of irreducibility.	of strangulation.			
1	F., 56	Unbilical	26 years	1 year	2 days	Small intestine, omentum	Adhesion of omentum and intestine to sac	Herniotomy Died. Fatty heart; pulmonary thrombosis.
2	F., 56	Right femoral	30 years	6 years	2 days	Small intestine, omentum	Adhesion of omentum and intestine to sac	Herniotomy Recovered.
3	M., 21	Left inguinal	All his life	Always	—	Omentum	Thickened and adherent omentum	Radical cure Recovered.
4	M., 63	Left inguinal	8 years	Several years	4 days	Small intestine, omentum	Thickened and adherent omentum	Herniotomy. Recovered.
5	M., 41	Right inguinal	Many years	Many years	—	Omentum	Omentum adherent to sac	Radical cure Recovered.
6	M., 67	Right inguinal	18 years	18 years	6 days	Small intestine	Coils of intestine adherent; band of adhesion across sac	Herniotomy Died. Collapse.
7	M., 45	Left inguinal	18 years	6 months	—	Omentum	Thickened and adherent omentum	Radical cure Recovered.
8	F., 39	Left femoral	Some years	Uncertain	—	Omentum	Thickened and adherent omentum	Radical cure Recovered.
9	F., 46	Left femoral	3 years	3 years	4 days	Omentum, small intestine	Thickened and adherent omentum	Herniotomy Died. Perforation of bowel.
10	F., 59	Right femoral	2 years	Uncertain	5 days	Omentum, small intestine	Adherent omentum	Herniotomy Recovered.
11	M., 36	Right inguinal	Uncertain	Uncertain	—	Omentum	Thickened and adherent omentum	Radical cure Recovered.
12	F., 42	Right femoral	4 years	4 years	—	Omentum	Omentum adherent to sac	Radical cure Recovered.

No.	Sex and age.	Situation of hernia.	Duration		Contents of sac.	Cause of irreducibility.	Treatment.	Result.
			of hernia.	of irreducibility.				
13	M., 59	Left inguinal	Many years	—	Omentum	Thickened and adherent omentum	Radical cure	Recovered.
14	M., 48	Left inguinal	Many years	Many years	Cecum and appendix and portion of small intestine	Doubtful	Herniotomy	Died 8th day. Collapse. No P.M.
15	M., 25	Left inguinal	2 months	24 hours	Small intestine	Intestine adherent to sac	Herniotomy	Recovered.
16	M., 41	Left femoral	15 years	Uncertain	Omentum	Thickened and adherent omentum	Radical cure	Recovered.
17	M., 40	Left inguinal	5 years	Some months	Omentum	Thickened and adherent omentum	Radical cure	Recovered.
18	M., 30	Left inguinal	Since birth	Uncertain	Omentum	Thickened and adherent omentum	Radical cure	Recovered.
19	M., 21	Right inguinal	4 years	4 years	Omentum, small intestine	Omentum adherent to sac	Herniotomy, radical cure	Recovered.
20	F., 85	Unilateral	14 years	Several years	Omentum, small intestine	Omentum adherent to sac	Herniotomy, radical cure	Recovered.
21	M., 36	Left inguinal	9 years	9 years	Omentum	Omentum adherent to sac	Radical cure	Recovered.
22	M., 30	Left inguinal	5 years	1 week	Omentum	Thickened and adherent omentum	Radical cure	Recovered.
23	F., 38	Right inguinal	4 years	4 years	Omentum	Omentum adherent to sac	Herniotomy, radical cure	Recovered.
24	M., 49	Left inguinal	18 years	Some months	Omentum, small intestine	Thickened and adherent omentum	Radical cure	Recovered.
25	M., 35	Right inguinal	5 years	5 years	Omentum	Omentum adherent to sac	Radical cure	Recovered.
26	M., 38	Right inguinal	10 years	Uncertain	Omentum and small intestine	Omentum adherent to sac	Herniotomy, radical cure	Recovered.

		femoral	4 years	4 years	—	Omentum	Thickened and adherent omentum	Omentum adherent to sac	Radical cure	Recovered.
31	F., 38	Left femoral	4 years	4 years	—	Omentum	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
32	F., 47	Un-bilical	16 years	16 years	2 days	Omentum, small intestine	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
33	M., 30	Right inguinal	1 year	1 year	—	Omentum	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
34	M., 52	Right inguinal	10 years	10 years	—	Omentum	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
35	M., 18	Left inguinal	Since birth	Always	—	Omentum	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
36	M., 46	Left inguinal	7 years	5 years	—	Omentum, small intestine	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
37	M., 29	Left inguinal	5 years	2 weeks	—	Omentum, small intestine	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
38	F., 52	Left femoral	Many years	20 years	24 hours	Omentum, small intestine	Omentum adherent to sac	Omentum adherent to sac	Herniotomy	Recovered.
39	M., 29	Right inguinal	7 years	Several years	3 hours	Small intestine	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
40	M., 23	Right inguinal	15 months	15 months	—	Omentum	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
41	F., 65	Right inguinal	30 years	1 week	—	Omentum	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
42	F., 37	—	2 years	6 months	—	Omentum	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
43	F., 51	Right femoral (left femoral reducible)	6 years	3 months	—	Omentum	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
44	M., 18	Right inguinal	Uncertain	Uncertain	—	Omentum	Omentum adherent to sac	Omentum adherent to sac	Radical cure	Recovered.
45	M., 50	Left inguinal	20 years	20 years	4 days	Omentum, small intestine	Omentum adherent to sac	Omentum adherent to sac	Herniotomy	Died an hour after operation.

No.	Sex and age	Situation of hernia	Duration		Contents of sac.	Cause of irreducibility	Treatment.	Result.
			of hernia.	of irreducibility, of strangulation.				
46	M., 22	Right inguinal	4 years	3 weeks	—	Uncertain	Radical cure	Recovered.
47	F., 74	Right femoral	40 years	4 days	Omentum and small intestine	Omentum adherent to sac and surrounding bowel	Herniotomy	Recovered.
48	F., 71	Left femoral	Many years	—	Omentum and small intestine	Thickened and adherent omentum	Radical cure	Died. Abscess from perforation of colon; broncho-pneumonia.
49	M., 52	Right inguinal	Many years	Uncertain	Small intestine	Intestine adherent to sac	Radical cure	Recovered.
50	M., 30	Right inguinal	16 years	—	Omentum	Thickened and adherent omentum	Radical cure	Recovered.
51	M., 42	Right inguinal	20 years	—	Omentum, small intestine	Omentum adherent to sac	Radical cure	Recovered.
52	F., 70	Right femoral	Many years	8 hours	Omentum, small intestine	Thickened and adherent omentum	Herniotomy	Recovered.
53	M., 32	Right inguinal	Many years	—	Small intestine	Intestine adherent to testis	Radical cure	Recovered.
54	M., 49	Left inguinal	5 years	—	Omentum, small intestine	Omentum and intestine both adherent to sac	Radical cure	Recovered.
55	M., 66	Right inguinal	8 years	1 year	Small intestine	Great thickening of mesentery	Radical cure	Recovered.
56	F., 38	Right inguinal	24 years	2 weeks	Small intestine, blood-clot	Sac thickened by laminated and adherent clot	Radical cure	Recovered.
57	M., 29	Right inguinal	7 years	6 months	Omentum	Omentum adherent to sac	Radical cure	Recovered.
58	M., 51	Right inguinal	30 years	20 years	Small intestine	Coils of intestine adherent to each other and adjacent to mesentery	Radical cure	Recovered.
59	M., 40	Right inguinal	Uncertain	Uncertain	Small intestine	Omentum adherent to sac	Radical cure	Recovered.

					hernia) Omentum	Thickened and adherent omentum	Radical cure	Recovered.
63	M., 34	Right inguinal	From childhood 6 years	—	Omentum and small intestine	Thickened and adherent omentum	Radical cure	Recovered.
64	F., 54	Right femoral	Many years	—	Small intestine	?	Radical cure	Recovered.
65	M., 40	Left inguinal	7 years	—	Omentum	Omentum adherent to sac	Radical cure	Recovered.
66	M., 25	Right inguinal	7 years	—	Omentum	Thickened and adherent omentum	Radical cure	Died. Old thickening of bowel; distension; fatty heart.
67	F., 43	Um- bilical	9 years	—	Omentum	Omentum adherent to sac	Radical cure	Recovered.
68	M., 62	Left inguinal	12 years	—	Omentum	Omentum adherent and thickened	Radical cure	Recovered.
69	F., 37	Right femoral	7 years	—	Omentum and small intestine	Omentum adherent to sac	Radical cure	Recovered.
70	M., 45	Left inguinal	20 years	—	Omentum	Omentum adherent to sac	Radical cure	Recovered.
71	M., 23	Left inguinal	6 months	—	Omentum	Omentum adherent to sac	Radical cure	Recovered.
72	M., 35	Left inguinal	Several years	—	Omentum	Omentum adherent to sac	Radical cure	Recovered.
73	M., 42	Ventral	3 months	—	Subperitoneal fat projecting through opening in recti	Fat spread out and indurated	Radical cure	Recovered.
74	F., 54	Right femoral	Several years	—	Omentum	Omentum nodular and adherent and containing cysts	Radical cure	Recovered.
75	F., 82	Left femoral	Uncer- tain	—	Omentum	Omentum adherent to sac	Radical cure	Recovered.
76	F., 65	Femoral	Uncer- tain	—	Omentum	Omentum adherent to sac	Radical cure	Recovered.

No.	Sex and age.	Situation of hernia.	Duration		Contents of sac.	Cause of irreducibility.	Treatment.	Result.
			of hernia.	of irreducibility.				
77	F., 60	Right femoral	1 year	—	Omentum	Omentum twisted and swollen	Radical cure	Recovered.
78	M., 54	Right inguinal	29 years	—	Small intestine	Coils of intestine adherent to each other and to sac	Radical cure	Died 5th day. Paralysis of intestine.
79	F., 24	Left femoral	Uncertain	—	Omentum	Omentum adherent to sac	Radical cure	Recovered.
80	F., 47	Unilateral	6 years	4 days	Cæcum and vermiform appendix; small intestine, omentum	Thickened and adherent omentum, and large size of hernia	Herniotomy	Recovered.
81	M., ?	Inguinal	Many years	? 2 days	Small intestine	Adhesion of coils of intestine	Herniotomy	Died 3rd day. No P.M.
82	M., 73	Right inguinal	Many years	1 day	Small intestine, blood-clot	Large amount of intestine in sac	—	A very fat, feeble man. Recovered.
83	F., 87	Unilateral	Uncertain	2 days	Omentum	Omentum adherent to sac, from which extended a band which caused internal strangulation of bowel	Herniotomy, abdominal section, division of band of adhesion	An enormously fat woman, 4 months pregnant. No intestine in sac, but internal strangulation by band. Abortion and death on 3rd day.
84	M., 56	Right inguinal	Several years	—	Omentum	Thickened and adherent omentum	Herniotomy, omentum removed	Died. Cerebral hæmorrhage. Alcoholic subject.
85	M., 48	Left inguinal	A year	—	Omentum	Thickened and adherent omentum	Radical cure	Recovered.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,'



DESCRIPTION OF PLATE XXVI.

On Irreducible Hernia (WARRINGTON HAWARD).

Case of very large Inguinal Hernia in a woman. The hernia consisted of bowel, and had been irreducible for more than a year, but no truss had been worn. It was entirely reduced in three weeks by the treatment described in the text.

(From a photograph by Mr. Clinton Dent.)



ON THE OCCURRENCE
OF
MUSICAL MITRAL MURMURS IN CONNECTION
WITH AORTIC STENOSIS

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A boy, Alfred S—, 19 years of age, came under my care at St. George's Hospital on the 24th of February, 1887. The previous history has no bearing on my present purpose. He was partially cyanosed and in a state of extreme cardiac distress; he had much dyspnœa, and was dropsical. The radial pulses were imperceptible, but the heart and carotids were beating violently and at the rate of 174 a minute. The impulse of the heart was extensive, and was attended with a force and sharpness which imparted to the hand a sense of great intra-cardiac tension, which impression was enhanced by the sharpness and accentuation of the sounds. The left ventricle seemed to be distended nearly to

bursting. The sounds were somewhat irregular, and there was an intense systolic murmur at the apex which had a musical character, or as the Medical Registrar, Dr. Slater, expressed it, "a high musical tone." By its intensity it gave the impression of being made under great force. This murmur culminated 2 inches interior to the nipple and 1 inch below it. It gradually diminished towards the aortic area, and was obviously mitral regurgitant. The urine was lithatic and albuminous, the liver enlarged to the touch. Bleeding was clearly indicated, and was practised from the arm to the amount of 6 ounces with advantage; the sounds of the heart became less intense, slower, and more regular. On the 1st of March there was slight hæmoptysis. Bronchitic râles presented themselves extensively, with orthopnoea, and he died seven days after admission.

On post-mortem examination the œdema was very conspicuous. The pericardium contained about 10 ounces of clear fluid. The heart was hypertrophied to the weight of 17 ounces. Both ventricles were dilated, the left greatly hypertrophied. The aortic orifice was almost completely closed by a mass of soft vegetations, the result, apparently, of endocarditis of no very remote date. These choked the passage so that no opening was visible; water passed from the ventricle to the aorta only by slow drops, from the aorta to the ventricle not at all. The mitral valve was practically healthy; upon it were a few small patches of atheroma. The tricuspid and pulmonary valves were perfectly healthy. Each pleura contained about a pint of fluid. The lungs were congested and œdematous; in the left was an apoplectic block. The liver was in a state of venous congestion, and weighed 4 pounds 4 ounces.

This case was, to me at least, instructive and suggestive. Though the disease was aortic obstructive, the murmur was mitral regurgitant, and some of the results—the pulmonary apoplexy, the enlargement of the liver, and the state of the urine—were characteristic of mitral regurgitation.

The distension of the left ventricle was declared by all its sounds and movements, and it could not but be inferred that the exit was in some way impeded. The closure of the aortic orifice, as exposed after death, was so nearly complete that it was not easy to imagine how the circulation could have been carried on. No doubt the great force of the hypertrophied ventricle sufficed to drive through more blood than the post-mortem condition appeared to allow of, though not enough to make a murmur. The only murmur was mitral, though the mitral valve was apparently efficient. As to the manner of its production, it can but be suggested that the valve leaked not because of any imperfection in itself, for there was practically none, but because of the repletion of the ventricle and the pressure within it.

The leakage occurred under extraordinary force, and was attended with a murmur of extraordinary tone. Thus a mitral murmur, and that a musical one, was the result of disease practically limited to the aortic valve.

The impression made on my mind by this case kept me on the look-out for others like it, some of which I will annex, selecting those only which were verified by post-mortem examination.

William C—, 38 years of age, came into St. George's under my care on the 27th of November, 1891. He had had rheumatic fever when young. He was dropsical and short of breath. He had evidence of great hypertrophy of the heart, and a murmur which was thought to be aortic diastolic, though there was some doubt as to its time. The second sound was defective. The pulse was small, not distinctly that of aortic regurgitation. There was little to note bearing on the matter in hand until the following March, when a musical apex murmur attracted attention. On the 7th of this month the condition was as follows:—The heart gave evidence of immense hypertrophy outwards and downwards, and had a tumbling rhythm. At the apex, $2\frac{1}{2}$ inches below and $1\frac{1}{2}$ inches outside the

nipple, was an ordinary blowing systolic murmur, with which was occasionally a definite musical sound or squeak, which was determined, on the joint evidence of several listeners, to resemble the squeak of a mouse. This appeared to be within the blowing murmur, but was quite different in tone. At the base no murmur could be recognised; the second sound was audible. The mitral squeak was held to indicate aortic stenosis. He died on March 15th, with the ordinary symptoms of valvular disease.

After death the heart weighed 27 ounces. The pericardium was much thickened; it contained calcified plates, and the two layers were everywhere firmly united. The left ventricle was greatly hypertrophied and slightly dilated. The left auricle was hypertrophied. The aortic valves were much deformed by fibroid thickening, especially about their surfaces of contact, and were so rigid as to narrow the channel and obstruct the passage. The mitral flaps were thickened, and the orifice crescentic and contracted, so as to admit only the tips of two fingers. On the auricular aspect of the valve were some small vegetations. The tricuspid orifice was dilated, the pulmonary natural. The right auricle was dilated; the right ventricle dilated and hypertrophied.

In this case there was, in addition to other murmurs, a squeak, which was musical if the cry of a mouse be so, which squeak was apparently made in the course of mitral regurgitation, and occurred in the presence of aortic stenosis. The sound was presumably imparted to the murmur by the intra-ventricular tension occasioned by the hypertrophy of the heart and the state of the aortic exit.

Charles C—, aged 69, was admitted into St. George's Hospital on June 12th, 1890. He was dropsical. He had shortness of breath, cough with blood-streaked expectoration, rhonchi over both lungs, and signs of emphysema. The cardiac dulness was increased, the

impulse not to be felt. The heart's sounds were much masked by sounds in the air-tubes, but on the evening of admission Mr. (now Dr.) Davidson, a very competent house physician, heard just to the left of the sternum, in the fifth interspace, a distinct whistle which was synchronous with the systole, and traceable to the axilla. During the night he became cyanosed and delirious, and in the morning died.

I need record only the state of the heart, which had attained the weight of 18 ounces. The left ventricle was hypertrophied and uncontracted. The aortic valves were calcified, adherent to each other, and the orifice in a condition of marked stenosis. The mitral valve was thickened, the orifice not dilated.

I have ventured to include this case though the death of the patient prevented any further observation than that recorded by Dr. Davidson. This however, is, I believe, to be relied on. As in the former instances, there was a musical murmur with the characters of mitral regurgitation, associated with aortic stenosis and hypertrophy of the left ventricle.

William H—, aged 54, was admitted into St. George's Hospital on the 7th of April, 1895, under Dr. Ewart, who permitted me to examine the patient. He had had several attacks of acute rheumatism. On listening to the heart attention was at once attracted by a distinct systolic squeak, like the high note of a fiddle, which was heard all about the apex, loudest $2\frac{1}{4}$ inches below the nipple and 1 inch to its inner side. The apex beat was indistinct, best seen on the level of the ensiform cartilage a little to its left. The first sound was scarcely audible, the second not at all. The dulness was increased, but somewhat indefinite. Pulsation was very visible at the episternal notch, not so marked in the carotids. The radial pulse was large, had a jerk suggestive of aortic regurgitation, and required hard pressure to stop it. The patient died on the day on which this note was made.

Post-mortem.—The heart weighed 17 ounces. Both ventricles were hypertrophied. The aortic orifice was obstructed by a luxuriant mass of solid vegetation which was attached to the edges of the valves, which themselves were rigid from old thickening and stuck out abruptly into the channel, so that partly from the state of the valves and largely from the vegetations upon them, the opening appeared to be almost closed. There was a patch of small vegetations on the septum just underneath the aortic valves. The mitral and tricuspid valves were comparatively little affected. The edges of the mitral were slightly thickened, and on its auricular aspect was a vegetation of the size of a small split pea. There was a little old nodular thickening on the edge of the tricuspid.

In this case, as in the others, there was a musical apex murmur, apparently mitral regurgitant, which was concurrent with, and probably dependent upon, aortic stenosis. The mitral valve was itself diseased, as in most of the other instances, but very slightly so. The conditions essential to the musical and intense murmur which this case and the others exemplify would seem to be mitral regurgitation in small volume at high pressure, the pressure being provided by hypertrophy of the ventricle and aortic obstruction.

Since my attention was drawn to the subject by the first case, that of Alfred S—, I have seen several marked instances, beside those related, in which a sound like the squeak of a small animal, a mouse or guinea-pig, thought to be a mitral regurgitant murmur, has been associated with signs of aortic stenosis; but as these clinical observations have not been explained by post-mortem examination, I have not included them in my series. The examples thus verified are few, and I should have preferred to wait for more had I any reasonable chance of obtaining them; but my hospital work has come to an end, and with it the probability of being able to add post-mortem evidence to what I have already adduced. There may be musical murmurs

of many kinds, as there are many sorts of musical instruments ; I speak only of one, and that one something of a paradox, a mitral murmur which owes its existence or its character to aortic disease.

(For report of the discussion on this paper, see ' Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 155.)

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CHARTER AND BYE-LAWS
OF
THE ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF
LONDON.



LONDON:

1897.



CHARTER

AND THE

BYE-LAWS,

AS APPROVED AND CONFIRMED BY A SPECIAL
GENERAL MEETING, JUNE 8TH, 1897.

CHARTER.

WILLIAM THE FOURTH, by the Grace of God of the United Kingdom of Great Britain and Ireland King, Defender of the Faith—To ALL to whom these presents shall come GREETING.

5 WHEREAS John Elliotson Doctor of Physic, Sir Astley Paston Cooper Baronet, and John Yelloly Doctor of Physic have, by their petition, humbly represented unto us that a Society was formed in the year one thousand eight hundred and five, by a considerable number of Physicians and Surgeons of eminence in London, for the cultivation and promotion of Physic and Surgery, and of the branches of Science connected with them, of which the last two named of the petitioners were original members; and that the said Society has expended considerable sums of money in the purchase and collection of a large and valuable library, and has published eighteen volumes of Transactions which have had a very extensive circulation. AND WHEREAS they, the said petitioners, have humbly besought us that we should give to them and to the other persons who have already become Members of the said Society, or who may at any time hereafter become Members of it, our Royal Charter of Incorporation for imparting greater stability and effect to the designs of the said Society. Now KNOW YE, that we, being desirous of encouraging a design so laudable, have and grant of it to the present Members and those who shall be elected according to future Bye-laws.

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I. Petition for a Charter,

Yelloly, and such others of our loving subjects as are now Members of the said Society, or who shall at any time hereafter become Members thereof, according to such By-laws as shall hereafter be framed or enacted, shall by virtue of these presents be called Fellows of the said Society, and shall be one body politic and corporate, by the name of *The Royal Medical and Chirurgical Society of London*; of which Society we do hereby declare ourselves, and successors if they shall think fit, the Patron, by which name they shall have perpetual succession, and a common seal with full power to alter, vary, break and renew the same at their discretion, and by the same name to sue and be sued, to implead and be impleaded, to answer and be answered unto in every court of us our heirs and successors, and be for ever able and capable in the law to purchase, receive, hold, possess, and enjoy to them and their successors, any goods and chattels whatsoever, and also be able and capable in the law (notwithstanding the Statutes of Mortmain) to take, purchase, hold and enjoy to them and their successors, any lands, tenements or hereditaments whatsoever, the yearly value of which shall not exceed in the whole the sum of two thousand pounds, computing the same respectively at the rack rent which might have been had or gotten for the same respectively at the time of the purchase or acquisition thereof; and shall have full power and authority to sell, alien, charge or otherwise dispose of any real or personal property so to be by them acquired as aforesaid, and to act and do in all things relating to the said corporation in as ample manner and form as any other our liege subjects, being persons able and capable in the law, or any other body politic and corporate in our said United Kingdom of Great Britain, may or can act or do.

II. Appointment of the first President, and first two Members of Council,

AND WE DO further declare and grant that for the better government of the said Society and for the better management of the concerns thereof, there shall be, from the date of these presents thenceforth and for ever, a President of the said Society, who with twenty Fellows to be elected in

- manner hereinafter mentioned shall form the Council. And we do hereby appoint the said John Elliotson the first President of the said Society, and the said Sir Astley Paston Cooper and John Yelloly the first Members of the Council,
- 5 to continue in office till the first day of March next. AND who are to be joined within four months by eighteen other Members of Council
- WE FURTHER DIRECT that within four months from the date of these letters patent, a General Meeting of the Fellows of the said Society shall be held, who shall be authorised by method of ballot to elect eighteen fit and proper persons as officers and other Members of the Council, Meeting: and to complete the number of twenty-one, of whom including all to remain in office till the 1st of March.
- 10 the President WE have willed that the Council shall be composed, and that such additional persons shall likewise continue in office till the first day of March next, and till
- 15 other fit and proper persons be chosen in their room.
- AND OUR further will and pleasure is, that the Fellows of the said Society shall and may on the first day of March one thousand eight hundred and thirty-five, and also shall and may on the first day of March in every succeeding
- 20 year or as near the same as conveniently may be, assemble together at the then last or other usual place of meeting of the said Society, and proceed by method of ballot to nominate and appoint a President of the said Society, and such Officers and other Members of the Council as may
- 25 with the President form the number of twenty-one, of whom we have willed that the Council shall consist; and also may, in case of the death, resignation or removal of the President or any Officer or other Member of the Council within the space of three months next after such death, resignation or
- 30 removal, elect some other person being a Fellow of the said Society to supply the place of such President or Officer or other Member of the Council. And Our further will and pleasure is, that no Fellow who has filled the office of President for two successive years shall be again eligible to
- 35 the same situation until the expiration of one year from the termination of his office, and that not more than two thirds of the Fellows who have formed the Council of the
- III. The Society to appoint twenty-one Officers and other Members of Council on the 1st of March in every year, o fill up vacancies that may occur in the intervals; not to appoint the same President for more than two successive years, nor ever elect more than two thirds of the

Members of the last Council; to have the power of electing new Fellows, and expelling Fellows.

The Council to have the power of making Bye-laws, for regulating the affairs of the Society, the description and number of its officers, elections and expulsions, appointment of subordinate Officers, filling up vacancies, determining the qualifications of candidates, the amount of entrance and subscription money, and the qualifications of Honorary Fellows; and to have the power of altering and revoking such Bye-laws as may be

preceding year shall be re-elected Members of the Council at such annual meeting. And we do further grant and declare that the Fellows of the said Society or any ten or more of them shall and may have power from time to time at the meetings of the said Society to be held at the usual place of meeting of the said Society, or at such a place as shall have in that behalf been appointed, by and with the consent of not less than four fifths of the Fellows present, to elect such persons to be Fellows of the said Society, and all Fellows to remove from the said Society, as they shall think fit; and that the Council hereby directed to be appointed and the Council of the said Society for the time being, or any three or more of them, all the members thereof having been first duly summoned to attend the meetings thereof, shall and may have power according to the best of their judgment and discretion to make and establish such Bye-laws as they shall deem proper and necessary for regulating the affairs of the said Society, and also the number and description of its Officers, and also the times, place and manner of electing and removing the Fellows of the said Society and all such subordinate servants, officers, and attendants as shall be deemed necessary or useful for the said Society, and also for filling up from time to time any vacancies which may happen by death, resignation, removal, or otherwise in any of the offices or appointments constituted or established for the execution of the business and concerns of the said Society, and for regulating and ascertaining the qualifications of persons to become Fellows of the said Society respectively, and also the sum and sums of money to be paid by them respectively, or any of them, whether upon admission or otherwise, towards carrying on the purposes of the said Society, and also the number, qualifications and privileges of such persons as they may from time to time deem it proper to admit as Honorary Fellows: and such Bye-laws from time to time to vary, alter or revoke and make such new and other Bye-laws as they shall think most useful and expedient, so that the same be

not repugnant to these presents, or to the laws of this our made,—
 Realm: PROVIDED, that no Bye-law hereafter to be made, ^{Provided}
 or alteration or repeal of any Bye-law which shall hereafter ^{the Society}
 have been established by the said Council hereby directed ^{sanction the}
 5 to be appointed, shall be considered to have passed and ^{proceeding of}
 be binding on the said Society, until such Bye-laws ^{the Council.}
 or such alteration or repeal of any Bye-laws shall, after
 such notice to the Fellows as from time to time may be
 deemed expedient by the said Society, have been confirmed
 10 by ballot by the members at large of the said Society, ten
 at least of the Fellows of the said Society being present:
 and Provided that no such Bye-law or alteration or repeal
 of any Bye-law shall be deemed or taken to pass in the
 affirmative, unless it shall appear upon such ballot that not
 15 less than two thirds of the Fellows present at such
 meeting shall have voted for the same. And Our further
 will and pleasure is, that it shall be lawful for any three
 Fellows, by writing under their hands transmitted to the ^{Any three}
 President or such other officer or officers as may by the ^{Fellows may}
 20 Bye-laws hereafter to be made be designated for the pur- ^{make recom-}
 pose, to recommend to the Council any new Bye-laws or ^{mendations}
 alteration or repeal of any existing Bye-laws; and in case ^{respecting}
 the Council shall not agree to such new Bye-laws or ^{Bye-laws to}
 alteration or repeal of any existing Bye-laws, then Our ^{the Council—}
 25 will and pleasure is, that such propositions shall, if ^{or to the}
 required by the said three Fellows, be submitted to the ^{Society, if}
 consideration of the Society at large, and determined on ^{the Council}
 by them in the same way as has been directed with ^{dissent from}
 regard to new Bye-laws or alterations or repeals of ^{the recom-}
 30 existing Bye-laws which have been approved by the ^{mendation.}
 Council. IN WITNESS whereof we have caused these
 Our Letters to be made Patent. WITNESS Ourselves,
 at our Palace at Westminster, this thirtieth day of
 September, in the fifth year of our reign.

35 BY WRIT OF PRIVY SEAL,

EDMUNDS.

BYE-LAWS.

CHAPTER I.

Of the Object and Constitution of the Society.

- Object of the Society.** I.—THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY is instituted for the cultivation and promotion of Medicine 5 and Surgery, and the branches of Science connected therewith.
- Number of Honorary Fellows.** II.—The Society shall consist of Fellows and Honorary Fellows. The Fellows shall be unlimited in number; the Honorary Fellows shall not exceed twelve British sub-10 jects, and twenty Foreigners.
- Radius of Resident Fellowship.** III.—Such of the Fellows as reside within seven miles of the Society's House shall be considered as Resident Fellows: all the others as Non-resident.
- Of whom Fellows may consist.** IV.—The Fellowship of the Society shall be restricted 15 to men, who are registered medical practitioners of the United Kingdom and to others whose qualifications are satisfactory to the Council.
- Honorary Fellows—British** V.—British subjects who have eminently distinguished themselves in Medicine, Surgery, or in Sciences con- 20 nected therewith, but are not engaged in practice, shall be eligible as Honorary Fellows.
- and Foreign.** VI.—Foreigners who have eminently distinguished themselves in Medicine, Surgery, or in Sciences connected therewith, shall be eligible as Honorary Fellows. 25
- Officers and Council.** VII.—The Officers of the Society shall be elected from the Fellows, and shall consist of a President, four Vice-

Presidents, two Treasurers, two Secretaries, and two Librarians, who together with as many other Fellows as shall make up twenty-one, shall constitute the Council, and shall have the management of the Society's affairs.

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CHAPTER II.

Of the Election and Admission of Fellows.

I.—EVERY Candidate for admission into the Society as Proposal of a Fellow shall be proposed and recommended by three or Candidates more Fellows, who shall deliver a paper signed by them- for Fellowship.

10 selves to one of the Secretaries, specifying the full name of such person, his professional qualifications and whence obtained, together with his department of practice and usual place of residence, and that he is a fit and proper person to be elected a Fellow; all which shall be certified
15 from their personal acquaintance with him. But, if such Candidate be resident abroad, such proposal and recommendation from one Fellow shall at the discretion of the Council be sufficient, together with a recommendation signed by two other Fellows, certifying, from their know-
20 ledge of his works, that he is a fit and proper person to be elected a Fellow. Every recommendation shall be Recommendations to be suspended in the Library of the Society for one meeting suspended in at least, exclusive of that on which it was presented, and the Library. shall remain suspended until the ballot for election shall
25 take place.

II.—A list shall be sent to each Resident Fellow List of fourteen days before the day of election, containing the Candidates to name of every Candidate whose recommendation has been be sent to suspended in accordance with the preceding law; and Resident
30 such list shall specify the full name of such person, his professional qualifications and whence obtained, together with his department of practice and usual place of resi-

Election by
ballot.

Votes
necessary to
secure
election.
When
elections take
place.

dence, and also the names of the Fellows who have recommended him to the Society for election. The election of Fellows into the Society shall be by ballot, and the ballot shall be taken in such manner as the Council shall from time to time determine; and no person shall be declared elected unless he have in his favour the votes of four fifths of the Fellows voting, ten Fellows at the least voting. The election of Fellows shall take place on the first Ordinary General Meetings of the Society in December, February, April, and June, and the ballot shall be taken in the presence of not less than two Members of the Council, and the result shall be declared by the President or Fellow presiding.

Appearing for
admission.

III.—Every person elected a Fellow of the Society shall, if he live within seven miles of the Society's House, have immediate notice of his election sent to him in such form as the Council may from time to time direct, and shall appear for his admission on or before the fourth Ordinary Meeting of the Society after his election, or within such further time as shall be granted by the Council; otherwise his election shall be void.

Obligation to
be subscribed
by Fellows.

IV.—Such person shall previously to his admission, subscribe the following Obligation in the Obligation Book:

“We, whose names are hereunto subscribed, having been elected Fellows of the Royal Medical and Chirurgical Society of London, hereby promise, that we will, so long as we shall be and continue Fellows, to the utmost of our power, promote the honour and interest of the said Society, and observe the enactments of its Charter and Bye-Laws.”

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If any person elected shall refuse to subscribe this Obligation, his election shall be void.

Form of
admission for
Resident
Fellows.

V.—The admission of any Fellow into the Society shall be at some meeting thereof, in manner and form following, he having first paid the Admission Fee and subscribed the Obligation; viz. being presented by some Fellow, the President, or Fellow officiating in his stead,

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shall address him in these words:—"By the authority and in the name of the Royal Medical and Chirurgical Society of London, I admit you a Fellow thereof."

VI.—Such persons as may be elected Fellows of the ^{Form of admission for Non-Resident} Society, and do not live within seven miles of the Society's ^{Fellows.} House, shall have immediate notice of their election sent to them, in such form as the Council may from time to time direct, together with an Obligation form, and shall be considered Fellows on paying the Admission Fee and ¹⁰ returning the Obligation duly signed on or before the fourth ordinary Meeting of the Society after their Election, or within such further time as shall be granted by the Council, otherwise their election shall be void.

VII.—The election, or re-election, of every person ^{Elections and re-elections to be recorded in a book.} into the Society, with the time thereof, shall be recorded in a book to be kept for that purpose; but if it appear ¹⁵ upon the ballot that the person proposed is not elected or re-elected a Fellow, no notice of the decision shall be taken in the minutes.

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CHAPTER III.

Of the Election of Honorary Fellows.

I.—THE power to recommend persons as ^{Recommendation and election of Honorary} Honorary Fellows shall be vested in the Council, and their election ^{Fellows.} shall be conducted in the same manner as that of other ²⁵ Fellows, with the exception that personal acquaintance shall not be necessary in recommending them.

II.—Honorary Fellows shall have a Diploma, in such ^{Form of admission for Honorary} form as the Council may from time to time direct, trans- ^{Fellows.} mitted to them; and when present at a General Meeting ³⁰ of the Society, shall be admitted with formalities similar to those prescribed for the admission of Fellows.

III.—The Honorary Fellows shall enjoy the privilege ^{Privileges of Honorary} of attending and introducing friends at the Ordinary ^{Fellows.} General Meetings of the Society, and when Resident, of .

using the Library in the same manner as ordinary Fellows, but shall not be entitled to any further privileges, without special leave, granted by the Society, at the recommendation of the Council.

CHAPTER IV.

5

Of the Withdrawing and Removal of Fellows.

Withdrawing
of Fellows.

I.—ANY Fellow may withdraw from the Society, upon signifying his desire to do so, by letter addressed to the Secretaries; provided he shall have paid whatever may be due from him to the Society, and shall have returned 10 all books in his possession belonging to the Society.

Removal of
Fellows.

II.—Whenever there shall appear cause, in the opinion of the Council, for the removal of any Fellow from the Society, a minute shall be made thereof, and a copy of such minute forthwith sent to the Fellow, and a special 15 meeting of the Council summoned thereupon, which the Fellow, whose conduct is in question, shall be invited to attend. If after investigation, the Council shall still consider the removal of such Fellow advisable, they shall frame a resolution to that effect, which shall be suspended in the 20 Society's Library for at least fourteen days, and be submitted to a Special General Meeting of the Society. On the resolution being put to the ballot, and four fifths of the Fellows present voting for it (ten at the least being present), the President or Fellow presiding shall declare 25 such Fellow removed from the Society accordingly.

CHAPTER V.

Of the Contributions of Fellows.

I.—EVERY person elected a Fellow of the Society, and living within seven miles of the Society's House, shall, previously to his admission, pay to the Society the sum of Six Guineas as an Admission Fee, which sum shall not be held to include an annual contribution; and shall afterwards contribute the sum of Three Guineas annually. But every Fellow may, after the payment of the Admission Fee, compound for his annual contribution by paying at one time the sum of Fifty Guineas. Every Fellow who has paid five or more annual contributions may compound for all future annual contributions, by paying at one time the sum of Forty-five Guineas; every Fellow who has paid ten or more annual contributions may compound for all future annual contributions by paying at one time the sum of Thirty-nine Guineas; every Fellow who has paid fifteen or more annual contributions may compound for all future annual contributions by paying at one time the sum of Twenty-two Guineas; every Fellow who has paid twenty or more annual contributions may compound for all future annual contributions by paying at one time the sum of Twenty-four Guineas; every Fellow who has paid twenty-five or more annual contributions may compound for all future annual contributions by paying at one time the sum of Fifteen Guineas; and every Fellow who has paid thirty or more annual contributions may compound for all future annual contributions by paying at one time the sum of Five Guineas: provided always that no Fellow may compound for future contributions, from whom an annual contribution is, at the time, due.

II.—All sums of money paid to the Society as composition fees in lieu of annual contributions shall be applied in

Admission Fee
for Resident
Fellows.

Annual
Contribution
of ditto.

Compounding
for the
Annual
Contribution.

How
Composition
Fees are to be
applied.

reducing the principal money owing upon the debenture loan of the Society, and shall be paid by the Treasurers to the Trustees for the debenture holders as and when the Council shall from time to time direct.

When yearly Contributions are payable.

III.—Annual contributions shall be payable at each 5 Annual General Meeting, for the current year; but no Fellow elected within three months preceding the Annual Meeting shall be liable to such contribution till the second Annual General Meeting from the time of his election.

Subscriptions in arrear.

IV.—Every Fellow whose annual contribution shall be 10 more than three months in arrear shall have his name suspended in the Society's Library, and shall not enjoy any of the privileges of a Fellow until such annual contribution be paid. If the said contribution shall not be paid on or before the first meeting in the November following, and 15 no reason be assigned, satisfactory to the President and Council, for such non-payment, he shall cease to be a Fellow of the Society. Provided that on a request in writing for readmission being addressed to the President and Council by an individual so circumstanced within the 20 space of three months following such meeting in November, the case shall be stated by the President from the Chair at one of the ordinary General Meetings of the Society, and the question of his readmission shall be decided by ballot at the next ordinary General Meeting; 25 but no person so applying shall be declared re-elected unless he shall have in his favour the votes of four fifths of the Fellows voting, ten Fellows at the least voting.

Admission Fee for Non-Resident Fellows.

V.—Every person who shall after the 8th day of June, 1897, be elected a Fellow of the Society and does not live 30 within seven miles of the Society's House, shall pay to the Society the sum of Three Guineas as an admission fee, and shall after the first year from his admission (the said admission fee being deemed to include his first year's annual contribution) contribute the sum of One Guinea 35 annually, and shall be entitled to receive the *Proceedings* of the Society, and to consult Books and Periodicals in

Annual Contribution of ditto.

the Reading Room, and to borrow one volume at a time from the Library. Any Non-resident Fellow wishing to enjoy the full privileges of a Resident Fellow, shall be at liberty to do so on paying an additional Three Guineas as admission fee and the annual contribution of Three Guineas.

5

How Non-Resident Fellows may enjoy the privileges of Resident Fellows.

VI.—Any Non-resident Fellow, who shall, by change of residence, become a Resident Fellow, shall subscribe the Obligation, pay an additional Three Guineas as admission fee, and the annual contribution of Three Guineas like other Resident Fellows.

Non-Resident Fellows who become Resident.

10

VII.—Any Resident Fellow of the Society, who by change of Residence shall become Non-resident, shall, from the date of the next Annual Meeting, be only liable to pay the annual contribution of a Non-resident Fellow: and the same shall apply to any temporary absence, provided it include the whole period between one Anniversary Meeting and another.

Resident Fellows who become Non-Resident.

15

VIII.—The determination of what constitutes Residence, or Non-residence, and the adjustment of any payment as a composition fee in lieu of annual contributions, or for the 'Transactions,' which may become necessary when a Resident Fellow becomes Non-resident, or when a Non-resident Fellow becomes Resident, shall be left to the discretion of the Council, as occasion arises.

25

CHAPTER VI.

Of the Election of Officers and Council.

I.—EVERY Fellow of the Society resident in the United Kingdom shall be summoned to the Annual Meeting, at least a week before the day on which it shall take place, by a letter signed by the President and the Secretaries.

Notices of the Annual Meeting sent to Fellows.

30

II.—All the Members of the Council shall be elected annually by ballot; but no Fellow shall be eligible to the

Election of Council and Officers.

Eligibility of
Fellows for
Offices.

office of President, Vice-President, or Councillor for more than two years in succession. Neither shall any Fellow be eligible for the office of Treasurer, Secretary, or Librarian, for more than two years in succession, unless recommended for re-election by a majority of not less than two thirds, 5 ascertained by ballot, in a meeting of Council specially summoned for the consideration of the house-list, provided always that not more than two thirds of the Fellows who have formed the Council of the preceding year shall be re-elected Members of the Council at such Annual Meeting. 10

Balloting lists
for Officers
and Council.

III.—Balloting lists, recommended by the Council, and having blank spaces for such alterations as any Fellow may wish to make in them, shall be laid on the Society's table, for the use of the Fellows, and sent to each Fellow resident in the United Kingdom, with the circular summons, 15 seven days previously to the day of election.

Ballot at the
Annual
Meeting.

IV.—The Chair shall be taken at the Annual Meeting at such time as shall be fixed upon by the Council, which time shall be inserted in the circular summons, and the ballot shall continue open for not less than one hour. 20

Scrutineers.

V.—The President, or Fellow presiding in his stead, shall appoint, from the Fellows present, two or more scrutineers, to superintend the ballot in its progress; and when it is closed to examine the lists, and report the result to the President. Each Fellow voting shall place his list, folded, 25 in the ballot box; and the name of each Fellow voting shall be recorded.

In case of
equality of
votes, election
determined by
President.

VI.—If any Fellows have an equal number of votes for an office in the Society, or place in the Council, the person to be elected shall be determined by the President. 30

CHAPTER VII.

Of the President and Vice-Presidents.

Duties of
President.

I.—THE President shall preside at all the Meetings, and regulate all the proceedings of the Society and Council.

He shall state and put questions, both in the affirmative and negative, according to the sense and intention of the Meeting; he shall maintain order in the proceedings, and execute, and see to the execution of the Provisions of the Charter and Bye-Laws of the Society. He shall, after the minutes of each meeting are read over, with the approbation of the Meeting, sign them, as a voucher for their accuracy.

II.—In the absence of the President, the Vice-Presidents ^{Duties of} 10 in rotation, or, in their absence, one of the Treasurers, or ^{Vice-Presidents or} a Fellow chosen by the Fellows present, shall take the ^{other} Chair, and act in all respects as the President is em- ^{Chairman.} powered to do by the Charter and Bye-Laws of the Society.

CHAPTER VIII.

15 *Of the Treasurers.*

I.—THE Treasurers, or some persons appointed by them, ^{Duties of} shall receive, for the use of the Society, all sums of money ^{Treasurers.} due or payable to the Society; and, out of such money, shall pay and disburse all sums of money which may be due 20 from, or payable by, the Society; and shall keep particular accounts of all such receipts and payments, in the way which may seem most proper to the Council.

II.—They shall not pay any sum of money on account of the Society without the sanction of the Council, or of a 25 Standing Committee, which has been empowered by the Council to order payments.

III.—All sums of money in the hands of the Treasurers, not immediately required for the use of the Society, shall be invested in such Government or other securities as shall 30 be approved of, and directed by, the Council.

IV.—The Treasurers shall keep a book of printed check receipts for annual contributions; each receipt shall be

signed by both of them, and be filled up with the name of the Fellow paying, the sum paid, and the time for which the contribution is paid. These receipts shall be countersigned by the person who shall receive the money on the Treasurers' behalf, and who, upon the delivery of the receipt to the Fellow paying, shall enter upon the counterpart thereof the above particulars, and the day of payment. The same proceedings shall be observed with regard to the receipts for admission and composition fees.

Accounts to
be audited
and laid before
the Council
and the
Society.

V.—The accounts of the Treasurers shall be audited annually by a Chartered Accountant to be appointed by the Council. The accounts so audited shall be laid before the Council at their meeting in February, and presented to the Society at the Annual General Meeting.

CHAPTER IX.

15

Of the Honorary Secretaries.

Duties of
Secretaries.

I.—THE Secretaries shall have the management of the correspondence of the Society and Council, except in so far as the Council shall otherwise direct.

II.—The Secretaries shall attend all meetings of the Society and Council, and shall be *ex officio* members of all Committees; when the Chair has been taken, one of them shall read the minutes, orders, and entries of the preceding meeting, and shall take minutes of the business and orders of the meeting; and at the meetings of the Society shall mention the gifts made since the last meeting; shall give notice of candidates that stand proposed for election into the Society; shall read the letters; and shall read the papers presented to the Society, as far as possible, in the order of time in which they were received, unless the President shall otherwise direct.

III.—The Secretaries shall have the charge, under the direction of the Council, of printing the Transactions and Proceedings of the Society, and of correcting the press.

CHAPTER X.

Of the Honorary Librarians.

I.—THE Librarians shall have the superintendence of ^{Duties of Librarians.} all matters relative to the Library, and be permanent members of the Library Committee.

II.—They shall, with the assistance of the Library Committee, inspect the Library once at least in every three years, and make a report on the state of it to the next Annual General Meeting of the Society.

10 III.—They shall, under the direction of the Council, order books, and be responsible for the printing of Catalogues of the Library, and for the entering of titles of all new works in the Catalogues.

CHAPTER XI.

Of the Resident Librarian.

I.—THE Resident Librarian shall either not be a Fellow ^{Resident Librarian and his duties.} of the Society; or, if a Fellow, shall cease to be so on his election to, and acceptance of, that Office.

20 II.—The Resident Librarian shall live in, and have the care of, the Society's house, and of the Library and other property contained in it; and shall give such security as may be required by the Council.

25 III.—He shall superintend all business connected with the Society's Library; issue the summons for all meetings, under the direction of the Secretaries; he shall always be in attendance at the meetings of the Society and of the Council; he shall be responsible for the collection of the annual contributions, and pay them to the Treasurers or persons whom they may appoint. He shall be subject

to such other rules and orders as may be given to him by the Council, and receive such remuneration for his services as they may deem proper.

IV.—He shall be in attendance during the hours in which the Library is open.

5

CHAPTER XII.

Of the Council.

Duties of the Council.

I.—THE Council shall have the management of the affairs of the Society, and shall appoint such resident officers and servants as they may deem necessary; shall 10 fix their duties, and suspend or remove them when they see occasion. They shall determine upon such security as may be proper to be given by such officers and servants.

Meetings of Council.

II.—The Council shall meet at the House of the Society at least once in every month, except in the months of July, 15 August, and September, or oftener should they see occasion; and three shall be a quorum, except in cases relating to the publication of Papers, when seven members must be present. A week's notice of each ordinary meeting shall be sent to every Member of the Council. Special Meetings 20 of the Council may be summoned by the President, or by any three Members.

Quorum of Council.

Special Meetings of Council.

Questions to be determined by vote or ballot.

III.—The following questions, when brought before the Council, shall be determined by ballot:—The election of officers and Members of Council. The appointment of 25 Referees. The acceptance and publication of Papers. All other questions in the Council shall be determined by vote, or by ballot if demanded; and in case of an equality of votes, the President shall have a second or casting vote; except in cases relating to the acceptance 30 or publication of a paper: when, if the votes be equal, the further consideration of the question shall be adjourned to the next Meeting of Council; and if on a second ballot

there shall still be an equality of votes, it shall be determined in the negative.

IV.—The Council shall determine upon the propriety of publishing such Papers as may be offered to the Society, ^{Publication of Papers.} 5 and also upon the place of their publication.

V.—The Council shall be empowered to appoint, at their first Meeting after the Annual General Meeting, ^{Referees to report on Papers.} thirty-six Fellows of the Society, as Referees, to report to them confidentially on the merits of Papers offered to the 10 Society, especially as to their fitness for publication in the Transactions of the Society.

VI.—The Council shall annually appoint a Library Com- ^{Library and House} mittee and a House Committee. They shall have it in their ^{Committees.} power to appoint as many other Committees as they may ^{Appointment of other Committees.} 15 think useful for promoting the objects of the Society, and to admit into such Committees any Fellows of the Society, whether Members of the Council or not. Such Committees shall act upon the instructions which they receive from time to time from the Council, to whom they shall report 20 their proceedings; and the appointment of all, except Scientific Committees, shall last for no longer time than up to the day of the succeeding Annual Meeting.

VII.—The Council shall exercise such other powers ^{General powers of the Council.} and authorities as are given to them by the Charter and 25 Bye-laws; and shall from time to time make such regulations and issue such orders, not inconsistent therewith, as shall appear to them conducive to the good government of the Society, and to the proper management of its concerns.

Of the Society's Transactions and Proceedings.

I.—THE Transactions of the Society, under the designation of MEDICO-CHIRURGICAL TRANSACTIONS, and the ^{Printing of Transactions.}

PROCEEDINGS shall be printed at such times and in such a manner as the Council shall direct.

Who are
entitled to
receive copies.

II.—Every Fellow of the Society paying an annual contribution of Three Guineas, whose annual contribution is not three months in arrear, shall be entitled to receive 5 one copy of each issue of the Transactions which may be published subsequently to his being admitted a Fellow; and the Council shall be empowered to present, in the name of the Society, copies of the Transactions and Proceedings to such scientific bodies as they may think 10 proper.

Presentation
copies.

Non-resident
Fellows may
compound for
Transactions.

III.—Every Non-resident Fellow of the Society, on payment of the sum of Eight Guineas, in addition to the usual admission fee, shall be entitled to receive one copy of every volume of the Society's Transactions which may 15 be published subsequently to such payment, provided his annual contribution be not more than three months in arrear.

Reprints of
papers
presented to
the authors.

IV.—Authors of Communications may, on application to the Secretaries, be furnished, at the expense of the 20 Society, with fifty copies of every paper presented by them, and printed in the Medico-Chirurgical Transactions: but such copies are not to be delivered to them, unless by a special order of the Council, till the volume, or part which contains the Paper, is ready for publication. 25

CHAPTER XIV.

Of Scientific Committees.

Appointment
of Scientific
Committees.

I.—THE Council shall, from time to time, appoint Committees of Fellows of the Society for the purpose of investigating questions of importance in Medical Science, 30 and shall have authority to grant such sums of money as they may deem necessary for the expenses of these investi-

gations. The Reports of such Committees shall be pre-Reports
 sented to the Council to be dealt with as may seem most published.
 advisable.

II.—Such Committees shall be called “Scientific Com-Constitution
 5 mittees.” They shall consist of not less than three Fel-of Scientific
 lows. The Secretaries of the Society shall, as far as Committees.
 possible, arrange that one of them shall take part in the
 work of such committees.

III.—The Scientific Committees shall continue to act Dissolution.
 10 until they have reported upon the subject referred to them,
 or been dissolved by the Council.

IV.—Every Member who has acted on a Scientific Com-Final Reports.
 mittee shall sign the final Report, or shall state, in writing,
 his reasons for declining to do so. The Report shall be
 15 first received and considered by the Council, specially
 summoned for that purpose, and shall afterwards, with
 their sanction, be presented at an Ordinary General Meet-
 ing of the Society. All Reports shall be placed in the
 Library at least one week before the day fixed for their
 20 presentation.

V.—The Council shall be informed by their Secretary Retirement of
 of the retirement of any Member of a Scientific Committee, Members.
 in which case the Council may, at their discretion, appoint
 another Fellow of the Society to fill the vacancy.

25 VI.—The names of the Fellows forming a Scientific Names of
 Committee shall be placed in the Library of the Society. Members
 placed in
 Library.

VII.—The Scientific Committees shall render to the All money to
 Council an account of all moneys received by them for be accounted
 for.
 the purposes of their investigations.

30 VIII.—The Council shall have the power to dissolve Power of
 a Scientific Committee whenever they may deem it Council to
 expedient. dissolve
 Committee.

CHAPTER XV.

Of the Library.

Management
of Library by
the Council.

I.—THE Library shall be under the management and direction of the Council, and be open on such days and at such hours as the Council shall direct. The Council shall be empowered to designate such works as shall not be allowed to circulate. 5

Borrowing of
books.

II.—Every Fellow paying the annual contribution of Three Guineas, or having compounded for the same, and every Resident Honorary Fellow, shall be allowed to borrow books from the Library, and to have eight volumes in his possession at the same time. Every Non-resident Fellow paying the annual contribution of One Guinea shall be allowed to borrow books from the Library, but shall not have more than one volume at a time. Pamphlets and periodical publications are not to be kept above one week, nor any other book above two weeks. 15

Number of
volumes and
time allowed.

Fines for
detention of
books beyond
stipulated
time.

III.—When a book is wanted which has been in the possession of a Fellow the stipulated time, the Resident Librarian shall send a notice by the post to the person in whose possession it may be, requesting the return of it; and a fine of Sixpence per day shall be incurred for every day that it may be detained, after the third from the transmission of such notice; and from the issue of such notice, and until the return of such work or works and the discharge of all fines incurred through delay, no further issue of books shall be permitted to such Fellow. 25

Books ordered
in for
inspection.

IV.—The books shall be ordered in for inspection at such times as the Council shall appoint, and a fine of Five Shillings per volume shall be incurred for neglecting to send in books by the time required in the notice.

Books
recommended
for purchase.

V.—A book shall lie on the Library table, in which Fellows may insert, for the consideration of the Council

or Library Committee, the titles of works which they think should be added to the Library.

VI.—Fellows who borrow books from the Library shall be answerable for the full value of any work that may be lost or injured while in their keeping. Responsibility of borrowers.

VII.—The Council shall have it in their power to collect the fines in the way which they may think best. Collection of fines.

CHAPTER XVI.

Of the Ordinary General Meetings.

- 10 I.—THE ordinary general meetings of the Society shall be held on the second and fourth Tuesday in the month, from the fourth Tuesday in October to the second Tuesday in June (both inclusive), at half-past eight o'clock in the evening. Meetings appointed for the
 15 election of Fellows shall commence at twenty-five minutes past eight o'clock. On the fourth Tuesday in December the Society shall not meet, nor on the usual Tuesday if it shall fall in Easter week, in which case there shall be a meeting on the fourth Tuesday in June. Ordinary General Meetings.
- 20 II.—Each Fellow of the Society shall have the privilege of introducing a stranger at every ordinary meeting, on delivering his name to the President or person acting in his stead; and the name of every stranger, so introduced, shall be entered in the Visitors' book; but no
 25 stranger shall be introduced more than three times in the same session. Introduction of strangers.
- III.—The business of the Society, at the ordinary meetings, shall be to read letters, reports and other papers on Medicine, Surgery, or any of the branches of
 30 Science connected therewith, and to converse upon professional subjects. Business at Ordinary Meetings.

IV.—At the ordinary general meetings of the Society,

nothing relating to its laws or management shall be brought forward.

Quorum.

V.—At ordinary general meetings, five shall be a quorum; but ten shall be necessary for the election of Fellows. 5

Additional Meetings.

VI.—Additional ordinary general meetings may be held when the Council may think them necessary.

CHAPTER XVII.

Of the Annual General, and Special General, Meetings.

Annual General Meeting on March 1st.

I.—THE Annual General Meeting of the Society, for the 10 election of the Officers and other Members of the Council, shall be held on the 1st of March unless that day shall happen to be Sunday, in which case it shall take place on the day following.

Special General Meetings.

II.—The President and Council may, at any time, call 15 a Special General Meeting of the Society, when it seems to them necessary; giving at least one week's notice, by letter, to every Fellow of the Society resident in the United Kingdom, of the time of meeting, and the business upon which it is summoned: and no business 20 shall be entered upon at such meeting except that which has been so notified.

Bye-laws—new proposals suspended in the Library.

III.—All proposals to enact, alter, or repeal, Bye-laws, shall be suspended in the Society's Library, for the inspection of the Fellows, from the time of the issuing of 25 the summons of the General Meeting, at which it is intended to submit such proposals, for confirmation by two thirds of the Fellows present, pursuant to the Charter.

Recommending new Bye-laws, and repealing or altering old ones.

IV.—Any three Fellows may recommend new Bye-laws, or the repeal or alteration of old ones, to the Council. 30 The recommendations, duly signed, must be sent in writing to one of the Secretaries. On such recommendations the Council shall deliberate at their next meeting; if the

decision shall not be satisfactory to the said three Fellows, the Council, if required by them, shall at the Annual General Meeting, or some Special General Meeting bring the same forward, with their decision thereupon, for the opinion of the Society at large.

V.—No resolution carried by way of original motion, or as an amendment, at the Annual General Meeting (except resolutions with respect to the election of the Council, and other prescribed matters, and the usual business of such meeting) or any other General Meeting, of which notice shall not have been given in the circular summoning such meeting, shall be binding on the Society or Council, until such resolution shall be confirmed by a Special General Meeting, to be convened within fifteen days of the Meeting at which such resolution shall have been carried, by notice from the Council, stating the object of such Meeting, and the resolution or resolutions to be proposed for confirmation, and such notice shall be sent to all the Fellows at least five days previous to such Meeting, and no question shall be discussed at such Meeting, of which notice shall not have been given in the summons.

Confirming
resolutions or
amendments
carried at
General
Meetings.

CHAPTER XVIII.

Of the Society's Property.

I.—THE whole of the Society's property and effects, of what kind soever, shall be under the direction, management, and control of the Council; but the Council shall not sell or mortgage any of the Society's lands, tenements, or hereditaments or invested funds without the sanction of a Special General Meeting of the Society or of the Annual General Meeting, due notice having been given of the business to be then taken into consideration.

II.—In order that this Society may in all respects conform with the provisions of the Statute 6 and 7 Vic., chap. 36, sec. 1, the funds of the Society shall at all times be

All funds to
be devoted to
the purposes
of the
Society.

devoted to the purposes for which it was instituted, and no dividend, gift, division, or bonus in money, shall at any time be made unto or between any of the Fellows.

CHAPTER XIX.

Of Donations to the Society.

5

Donors' names to be entered on the minutes, and

I.—THE name of every person who shall present books, money, or any other property, to the Society, shall be entered on the minutes, with the mention of the gift; it shall be announced at an ordinary meeting, or the Annual General Meeting of the Society, and inserted in the next 10 number of the Proceedings of the Society.

inserted in books presented.

II.—Books presented to the Society shall have the Donor's name inserted in them.

CHAPTER XX.

Of the Common Seal and Deeds.

15

The Common Seal of the Society.

I.—THE Common Seal of the Society shall be a representation of Salus raising a kneeling figure;* with the motto, NON EST VIVERE, SED VALERE, VITA; † and the date of the formation of the Society, 1805; surrounded by a Garter, having on it, SIG. SOC. REG. MED. CHIR. LOND. 20

Custody of the Charter, Seal, and Deeds.

II.—The Charter, the Common Seal, and the Deeds of the Society shall be kept in safe custody in such manner as the Council shall from time to time direct.

Use of Common Seal.

III.—The Common Seal shall not be affixed to any deed or writing, except at a meeting of the Council, and by 25 their authority.

* *Figura Muliebris stans; dextra, figuram virilem procumbentem sublevat; sinistra, baculum, serpente involutum, gerit.*—From a rare medal of Caracalla, in the British Museum, described in Vaillant's *Numismata Imperatorum Romanorum*. 30

An engraving of the Society's Seal is given on the title-page.

† Martial, Epigram. Lib. 6, Ep. 70.

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N.B.—The larger figure indicates the page, and the smaller figure
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